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SCARLATINA.[1]

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FOR many years past the case mortality rate of scarlatina in this country and in Great Britain and other European countries on the Atlantic seaboard has remained at a very low figure, generally about 1% or 2%. On the other hand, the case incidence in these various communities has not diminished. Naturally, there has been a good deal of questioning during the last few years as to whether, in view of the large expenditure of public money on the isolation and treatment of scarlatinal patients, we are not now taking the disease too seriously, and whether the continuance of such expenditure on the present scale is warranted. Commissions have sat, *questionnaires* have been sent to all parts of the world, reports have been issued and local experiments tried. While it should be admitted that the opinions given have been too much influenced by the statistician who, with his eyes glued to the consistently low mortality rate, has not paid sufficient attention to other clinical features, nevertheless the question is a very real one, and merits the closest study. The present paper is not meant as an expression of opinion one way or the other. It is merely a statement of present local conditions, the experience of the past will be brought in to guide us, an attempt will be made to discover if in fact the disease is worth worrying about, and whether it be so or not, to suggest means of lessening the invalidity and waste that it entails.

No one knows how long scarlatina has existed; one thing is certain, that it flourished long before it was recognized as a separate disease. Sydenham is generally credited with the first description, but it was known long before his time, and long after

[1] Read at a meeting of the Victorian Branch of the British Medical Association on August 5, 1931.

his death was still confused with measles, and right up to the nineteenth century with diphtheria. Its particular habitat has always been north-western Europe and North America, and its particular *penchant* the Anglo-Saxon race. If Sydenham had known how grievously England was to suffer, he would never, when asked concerning his methods of treatment, have contemptuously described it as "hardly worth the name of disease." [*Simplici hac et naturali plane methodo, hoc morbi nomen (vix enim altius assurgit) sine molestia, aut periculo quovis facillime abigitur.*]

So far as we know, the disease remained of a fairly mild character till about 1740, then there were increasing waves of virulence till the end of the century, when it died down both in Britain and America. From that date to the present day the history is practically complete; we can collect our facts and learn our lesson.

Taking the progress of the disease in Britain, after a premonitory rumble in 1825-1826 (note that there had been peace for a quarter of a century) the storm broke in 1832, and from then on till the late seventies there was little or no intermission, and Britain lost hundreds of thousands of lives. The end of the storm was as sudden and dramatic as it is to this day inexplicable, and since that time no severe epidemic of scarlatina has occurred in Britain.

The disease made its first appearance in this country about 1850, and following serious epidemics in Victoria in 1861 and South Australia in 1868, the terrible period of 1870-1876 followed, affecting the whole of southern Australia. On a certain day in 1876 prayers were offered up in the Melbourne churches for deliverance. Since 1880 no fatal epidemic of scarlatina has occurred in any Australian State.

Practically simultaneously with Great Britain, other north-western European States were attacked. Germany and Denmark were early sufferers; wide territories of western Russia were affected by 1835, and after thirty years of comparative quiet, malignant scarlatina broke out in North America in 1851. Scandinavia was affected later. A few figures will indicate the influence of scarlatina on the general mortality. In Norway, between the years 1867-1878, scarlatina accounted for 6·6% of all deaths. In 1876 one out of every eight deaths in Norway was from this disease. In London, from 1862 to 1865, 4·2% of all deaths were due to scarlatina.

This was the great wave on which attention has been focused, and on superficial study one would say that scarlatina as a malignant disease died out fifty years ago. And that is certainly the impression that one would gain from the medical literature of the past generation. But how does it stand in relation to the facts? On looking into the matter a little more deeply, one is led to the suspicion that for the past two centuries at least the world has never been free from malignant and widespread epidemics of scarlatina for any lengthy period.

Taking the quiet period of the first thirty years of last century, we find that Ireland experienced a malignant epidemic from 1801 to 1804, then after twenty-seven years of peace shared in the great outbreak of 1832. The disease appeared in a severe form in France in 1824 and swept across that country, Switzerland and northern Italy. Bretonneau, who for twenty-five years had thought scarlatina "nothing to worry about," woke up suddenly in 1825 and altered his opinion. Coming to the second quiet period, from 1878 onwards, we find that in middle Europe the violence of the disease died down much more slowly than in the countries of the Atlantic littoral, and that latterly a considerable rise of virulence has occurred all over eastern and south-astern Europe. The Balkan and Jugo-Slavonic States and Soviet Russia have suffered severely of late years. In isolated outbreaks in Roumania the case mortality has risen to over 90%. A fairly typical example is that of the town of Perm, in Soviet Russia. The total child population in 1927 was 23,086. During the years 1925-1928 there were 2,468 cases of scarlatina in children. Deaths numbered 343, representing a case mortality of 14%.

The conclusion to which we are led, I think, is that at any given time during the past two centuries scarlatina has been present in more or less malignant form in some part of the civilized world.

Nevertheless, it will be said, fifty years is a long time, and there have been fifty years of freedom from catastrophic epidemics in Britain and other Nordic countries and, for that matter, in Australia. Moreover, during that time the trend has been on the whole towards a lower and lower mortality. Are we not justified in hoping that this signifies the fixing of an endemic mild type of scarlatina and possibly its ultimate disappearance? It has been suggested that an immunity is gradually developing, following the great visitations of the nineteenth century, and in support of this there has been brought forward the greater proportionate incidence in older persons and the more pronounced immunity of children under one year. It may or may not be that there is sufficient evidence to justify a confident hope; to my mind we have no reasonable assurance in the matter.

Let us take the case of smallpox and alastrim for a comparison. Alastrim is a variant of smallpox. It has been endemic for years. Whether it is a fixed stable variant, or whether it may suddenly or gradually break into true smallpox is not known; at any rate there has been no tendency to date for it to do so. So far, therefore, it has been a fixed variant, and it may continue so. "Very well, then," say the optimists, "all we have to do is to keep Asiatic smallpox out and we are all right." And so they are.

But when we apply the comparison to scarlatina it breaks down at once. Modern scarlatina is not a fixed variant of anything. Alastrim breeds alastrim; every case of alastrim is a case of mild, non-fatal smallpox. By no means can it be said that

every case of scarlatina is a mild, non-fatal case of scarlatina. This naturally leads us to examine the various types of scarlatina as they exist here today, and see if they really are very different from those of the dreadful epidemics of sixty years ago.

First, let us consider those acute fulminating cases which in the old days always, and nowadays usually, result in death within three days or so. Such cases have for generations past been classed as (i) toxic or (ii) septic, and for convenient clinical description there is no reason why this classification should not be continued. It is obvious that in severe septic cases there will certainly be much toxæmia, and in many severe toxic cases streptococci will before death have had time to invade the blood stream, but in extreme and typical cases the distinction so far as clinical signs and *post mortem* appearances go, is fairly sharply marked.

The criterion of the "toxic" case is the fact that the patient is poisoned. Necrosis and even suppuration in the throat may be present, but there must be no secondary suppuration elsewhere. The short course of the disease varies somewhat, of course, in clinical details, but on the whole is surprisingly constant. In most instances, for the first few hours after an unimpressive onset there are no symptoms to cause great anxiety. Vomiting at the onset is usual in children, and also, in my small experience of this condition, diarrhœa. The soreness of the throat may be slight, moderate or great, but in any case the patient does not appear seriously ill. After six, twelve or possibly twenty-four hours have elapsed, there is a distinct and rapid change for the worse. Convulsions may occur at this stage, or respiratory distress with cyanosis. These, with a tendency to dulness or stupor, constitute this rapid change for the worse. A few hours later the patient is dead, generally between twenty-four and forty-eight hours from the onset. Death is painless, the stupor has passed into coma before the end. The temperature presents a steady rise till shortly before death, when it may rise very high or drop below normal.

Post mortem examination discloses variable minor changes in the heart and kidneys, and also in the lymph glands and lymphoid tissue throughout the body, with a tendency to small hæmorrhages. The most pronounced changes are found in the brain, in the throat of course, and, curiously enough, in the bronchi. The brain simply shows œdema, softening and small hæmorrhages. The bronchi and even the smaller tubes are intensely red and inflamed, with a tendency to hæmorrhages. The condition of the brain is the principal factor in the early death.

Patients belonging to this class present a wonderful opportunity for treatment by intravenous injection of antiscarlatinal (antitoxic) serum. This is the only treatment offering any chance of success. Should a visitation ever come upon us, I look forward to some dramatic recoveries. I note, however, that in Poland and in Roumania some disappointing results are being obtained in hypertoxic cases, and serum from convalescents is being given another trial. I am not particularly impressed with the prospects.

Illness of this kind is a rarity in Australia; I do not think I have seen more than three cases in the past ten years, and not many more in the preceding ten years. In 1916 one patient, whose symptoms very nearly justified his inclusion, recovered. In no other instance have I ever seen a patient live longer than two days.

The second type of fulminating case is the "septic." This is rather more common, though still fortunately a rarity. Of course, in many instances a patient dies of sepsis a week, a fortnight, a month or more after the onset of illness; such illnesses do not enter into this class. In the malignant septic case there is usually considerable toxæmia, and for the first twenty-four hours or even longer the course of the symptoms may be very similar to that in the toxic type. By this time, however, there are generally present sanious or serous rhinorrhœa, great swelling of the cervical glands and interglandular tissues, and also of the throat and fauces, with membrane formation, gross ulceration, or free pus flowing from the tonsils. The next day the nasal discharge is more profuse, the neck swelling is hard, board-like and enormous, hæmorrhagic spots or patches are likely to appear on the skin, the temperature assumes a swinging type, later becoming irregular, and death occurs, again quietly. In my experience death occurs nearly always in from two to four days from the onset. Prior to death signs of septic bronchitis or bronchopneumonia are nearly always present, the spleen is easily felt, and there is profuse diarrhœa. The patient does not live long enough for gross suppuration or gangrene to occur in the cervical swelling. I have noted little pockets of pus more than once, however, *post mortem*. The whole of the pharyngeal organs are found to be in a state of suppuration or necrosis, the bronchi and bronchioles are plugged with pus here and there, the spleen is large and soft, and minor signs are present in the other organs.

Now these two types of malignant infection were responsible, I think, for the majority of the deaths from scarlatina in the great epidemics of last century. Nowadays they account for a small minority. These toxic and septic forms occur today just as severely and kill just as surely and as rapidly as they ever did. But they occur very much less frequently. Moreover, I think that if malignant scarlatina does reappear in epidemic form, we shall by modern methods of treatment be able to reduce the mortality considerably in borderline cases. I believe that even in the malignant septic cases, if patients are seen reasonably early, there will be a chance of recovery if large doses of antitoxic serum be administered intravenously.

Leaving, then, the matter of deaths from fulminating infections, we have still to consider the fact that many patients died in later stages of the disease. What did they die of?

First I would put prolonged toxæmia and prolonged sepsis, the "typhoidal" type of scarlatina or the ordinary septic and pyæmic variety which we have with us today and which is the most frequently fatal type of case. Secondly, in the old days, diphtheria, without a doubt. Nowadays this danger does not exist. I have not seen a fatal case of post-scarlatinal diphtheria for very many years, and only one or two in which diphtheria was concurrent with scarlatina. Thirdly, in the old days, nephritis, early and late. This matter will be discussed later, but early nephritis does not often occur nowadays, and late nephritis, though common enough, is not often fatal. Bronchopneumonia and septic meningitis following otitis were probably the only two important remaining causes of death.

So now we have arrived at this position. If the worst comes to the worst, we are in a better position, as regards the saving of life, than our grandfathers were. Many of the patients whose cases approach the fulminating types as regards severity, may recover after energetic antitoxic treatment; we need not worry very much about diphtheria as a complication; and we should be able to make a better show as regards treatment of *otitis media* and nephritis, to name two of the fairly common causes of death. On the other hand, should the catastrophe not appear, we can jog along fairly comfortably, accepting our steady mortality rate of 1% or less, and the statisticians will shake their heads and say that really we are taking this minor ailment too seriously.

But can we just "jog along" like this? Let us look at the other side of the picture, which presents another story, of the really unsatisfactory features of the business.

Many of you probably still have the impression which I had myself, that scarlatina in the middle of last century was almost invariably a serious or at any rate fairly severe disease. That those who got it very badly, died, that some of the others died, and that the remainder had at any rate a nasty illness, most of them being left with deafness, otorrhœa, rheumatism, endocarditis or what not. As a matter of fact, this is stated almost in so many words in one of the recent reports to which I have alluded. In its worst periods the disease was not, and never has been, anything of the sort, as we shall see.

Let us now consider and compare those grades or varieties in which the immediate indication is not to save the patient from imminent death, but to minimize, if possible, any further period of poisoning, to prevent the further development or spread of septic processes, and to avoid relapse and reinfection. They include: (i) Patients with considerable toxæmia, and the normal angina of fairly severe scarlatina. (ii) Patients with gross local infection, involving not only the throat and nasal passages, but also the cervical lymph glands and tissues and often at an early stage the middle ear. (iii) Patients in whom the classical signs and symptoms are well marked, but without the serious conditions indicated in the first two classes. These present what may be called the moderate forms of scarlatina, which merge indefinitely into the well known mild form in which there is definite angina, a typical exanthem and fever for a few days only. (iv) Patients in whom the rash is absent or transient and the early symptoms slight. These four classes represent what we now regard as everyday scarlatina.

Now all these grades of severity in individual cases were present, not rarely, but very commonly, in the severe epidemics of last century. I am inclined to think that the first two grades of severe infection were proportionately more common then than now. Concerning the still milder forms I do not think it is possible to form a definite opinion, but one thing is certain, they were very common. The literature of the eighteen twenties and thirties right down to the nineties is full of references to them. I do not intend to weary you with long lists of references, but from Bretonneau and Trousseau right down through the long line of British, Norwegian, Danish and German observers, men who really did know something about scarlatina, one constantly finds references to groups of cases, occurring in the midst of most severe and fatal epidemics, in which the disease was mild and uneventful. Regarding *scarlatina sine exanthemate*, these words were written last century: "Without question, according to the unanimous opinion of the profession, people who suffer merely from angina, and have no exanthem, can transmit scarlatina." This is the mildest and most conservative pronouncement on the matter that I have read. The commonness of the condition was well recognized and insisted on, and certain "epidemics" of nephritis were ascribed by some, I believe correctly, to this cause. The significance of their statement will, I hope, become more apparent shortly.

Let us now for a time compare the symptoms and effects of these types of scarlatina sixty years ago and today. Leaving for the moment those cases of the mildest nature, there are one or two points of interest as regards the moderately severe forms. I doubt whether middle ear suppuration was any more common than it is today, and suspect that it was not on the whole more severe. Deafness appears to have followed more often than nowadays, but merely because the severer types were more common. The milder forms of scarlatinal rheumatism are just as common today in children, and because of its great frequency in adults and older children the total incidence of this symptom is much higher than it was. For a long time undue emphasis was placed on the "early nephritis" occurring in the first and second week, but after the description by Klebs in 1876 of his "glomerulo-nephritis," and the acrimonious discussions following it, "early nephritis" was considered simply as the immediate effect of a severe infection and similar to that which occurs in other diseases. We see it today in the severer types of case, but it does not occur, and never did occur, in mild scarlatina to any extent.

Scarlatina is the result of an infection of the throat by a streptococcus. A toxin is formed which produces an enanthem of the neighbouring mucous membranes, and shortly after a general exanthem affecting the trunk and limbs. If toxin absorption is sufficient, there may occur the usual effects on heart muscle, kidney tissues *et cetera*. This factor may be easily controlled in all moderately severe cases of scarlatina, by the early administration of antitoxic serum. The fever drops rapidly, the rashes fade, and the organic toxic effects simply do not occur. In these respects the action of and the results obtained by the use of antitoxic serum correspond exactly with those in diphtheria by its antiserum. Scarlatinal toxin is not nearly so potent as that of diphtheria, and a far larger proportion of patients not treated with serum recover without toxic complications than would be the case in diphtheria. In practically all early cases of scarlatina, so far as patients treated in hospital are concerned, the danger of immediate toxic effects can be ignored.

But there is another series of effects that cannot be ignored. The enanthem on the throat, tongue, palate, and nasal passages *et cetera* desquamates, leaving a raw surface. As virulent streptococci are on the spot, all the necessary factors are present for rapid extension of infection. The commonest local infections are those of the middle ear and of the cervical lymph glands, and minor ulcerative processes in the nasal passages, on the tongue, the palate, and the tonsils are common. The frequency of *otitis media* is partly due to the fact that the nasal passages cannot be efficiently cleansed or drained.

Now, if serum be given early, before desquamation of the enanthem has been completed, it is very unlikely that any such extensions of the infective process will occur. An occasional instance of *otitis media* occurs; in such cases I have more often than not found it to be merely a lighting up of an old ear suppuration. Theoretically, then, the early use of serum is justified not only by virtue of its neutralizing toxin and aborting the toxic symptoms, but also as a means of preventing septic complications in consequence of aborting the enanthem. Incidentally it may be said that it aborts the exanthem also; if it is given early, while the rash is yet young, it will fade rapidly and there will be no desquamation.

On the other hand, should serum administration be delayed for some days, not only is it too late to prevent toxic effects, but the enanthem has done its worst. Of course, this worst may not be very bad, and careful treatment and nursing will prevent any immediate extensions, but in the severer forms, otitis, adenitis and severe throat inflammation follow as a matter of course. In these cases localized suppurative adenitis or enormous cervical angina may occur, and pyæmia or septic bronchopneumonia is a recognized final development.

There is just one other early manifestation, which usually means trouble ahead. In many children, during the first two or three weeks of the disease, there is a tendency to irregular fever and persistently swollen glands in the neck. The glands are hard and discrete, tender and more swollen for a few days, corresponding with a rise in temperature, then partially subsiding and coming up again a few days later. These conditions occur in one class of children only, children with unhealthy, generally much enlarged tonsils, and who almost invariably give a history of previous similar trouble.

To summarize, then:

1. If serum is given early, then, whether the case is severe or mild, the immediate issue is the same; the primary disease is over in a few days at most, with rare exceptions.
2. If serum is not given early and the enanthem has been severe, local complications, such as *otitis media* and cervical adenitis, are to be looked for. If the enanthem has not been severe, most children escape all complications at this stage.
3. In children of a certain type, whether serum be given or not, and whether the scarlatina be severe or mild, adenitis of a variable or recurrent character is to be noted, and its presence is to be taken as a warning.

So much for the first period of the disease which, as a general rule, may be said to end about the eighteenth day. The second period possesses no great significance for the majority of scarlatinal patients, nearly all adults, most older children, and not a few young children go ahead unconcernedly, no complications occur, and after an uneventful stay they are discharged. For the remainder this second period is a time not only of ill-health, but of actual danger.

The first danger that comes to mind is, of course, that of nephritis. The initial mildness or severity of the scarlatina gives us no indication as to the likelihood of subsequent nephritis. To say that the vast majority of instances of nephritis coming under my notice have followed mild scarlatina is simply another way of saying that most of the scarlatina I have seen has been initially mild. The fact that mild scarlatina is more likely to be missed and the subjects not kept in bed and looked after, and developing nephritis possibly as a result, is counterbalanced by the fact that in severe scarlatina there is more early kidney damage and consequently increased liability later on.

For many years I have taught students that scarlatinal nephritis makes its appearance as a rule between the eighteenth and the twenty-third day. As this rather dogmatic and seemingly unreasonable statement was commented on adversely, I have naturally since then given the matter quite close attention and see no reason to alter the statement. For many years then, in the great majority of all cases of nephritis coming under my notice, the signs and symptoms began between those dates.

I do not intend to discuss nephritis in detail, but merely state that commonly its appearance is heralded by a slight or moderate, but definite rise

in temperature, and an equally definite swelling of the cervical lymph glands. To go a step further, should these two signs occur at the time stated, an immediate microscopical examination should be made of the urine, even if, as is commonly the case, this had been found normal a day or two before.

The onset of true scarlatinal rheumatism belongs also to the second period. It is true, of course, that rheumatism is common during the first week of the disease, usually of a mild or moderate character, but the true and typical scarlatinal rheumatism appears in the third week or later, and is accompanied by the same rise in temperature, the same tonsillar inflammation, and very often the same adenitis. I have seen children escape the rheumatism on the first and even the second occasion of this lighting up of infection, only to develop multiple articular rheumatism on the third occasion. Endocarditis and pericarditis may follow and death may occur quite quickly.

This brings us to the third manifestation of this second period, the development of a true "relapse," with angina, fever, enanthem and exanthem. In most text books and writings relapse is described as rare, very rare, or exceedingly rare; with the last pronouncement I am quite unable to agree. A relapse rate of even so little as 0·5% in a disease as common as scarlatina indicates a large number of actual instances. To justify including any case as one of relapse, it is necessary, of course, that there shall be no question as to the correctness of the diagnosis of scarlatina in the primary attack. Therefore, in my own records I ignore all cases in which the original diagnosis is open to doubt. There remains quite a goodly number of genuine instances of relapse. Prior to 1927 the incidence was small, never, I think, reaching 1% in any one year. Since then, however, there has been a very great increase. I commented on this at the Sydney session of Congress two years ago, and the increase has continued since. I suggested at the time that this was correlated with the use of antiscarlatinal antitoxin, but I think we shall find that this is only a part of a much larger and more comprehensive proposition. Ever since then we at Fairfield have been watching the matter very closely and looking out for confirmatory news from other countries. Two reports have been noticed during the past few months; Lichtenstein, of Stockholm, reports an increase last year to 10% of relapses, a higher figure than we have ever had in this country. The actual figures of classical "relapses," however, do not tell the whole story, for after all what is relapse?

In one respect at any rate, scarlatina is strictly comparable with diphtheria.

In diphtheria we have a tonsillar infection, toxin is diffused out. If this is neutralized, nothing happens; if not, membrane is formed. If sufficient toxin is absorbed before neutralization occurs, paralysis supervenes or toxic myocarditis. In scarlatina we have also a tonsillar infection, and toxin is elaborated. If it is neutralized, again nothing happens; if not, an enanthem appears, and if free toxin in sufficient amount is absorbed into the circulation, a generalized exanthem. We do not need the existence of paralysis to prove the existence of diphtheria, and we do not need the existence of the exanthem to prove the existence of scarlatina. Strictly speaking, therefore, a relapse may be taken to mean any lighting up of infection, or any lighting up of toxin production, to such an extent as to produce a lighting up or recurrence of symptoms. For many years, long before the scarlatinal toxin was shown to be streptococcal, I taught students that scarlatinal nephritis, for example, was to be regarded in the nature of a relapse, of a fresh outpouring of toxin through a kidney possibly damaged by the primary attack. Taking relapse, then, in its broadest sense, we may attempt its explanation by considering two hypotheses.

1. Patients lose their antitoxic immunity, either wholly or in part, by the end of two or three weeks. Streptococci remain in the local foci of infection, and shortly a complete or partial picture of the disease is reproduced.

2. Patients are infected by fresh strains of streptococci, to which and to whose toxins they have developed either no immunity, or only a partial one.

There are at least two other possible hypotheses, but they are too speculative to be included here. The first introduces the question of some factor additional to the streptococcus being necessary for the production of scarlatina. This might be a filtrable virus, or another streptococcus, or some quite intangible thing, a mere quality. The other involves the question of the mutation of streptococci, which, however important and far reaching, is as yet too indefinitely determined.

Let us now examine the two hypotheses and see if either of them will explain, to the exclusion of the other, the common symptoms and complications of the second period of the disease. Before doing this, it is necessary to enumerate and describe briefly these symptoms and complications. First of all of course, there is nephritis, accompanied very frequently indeed by cervical adenitis, a moderate tonsillitis, and slight or moderate fever. Scarlatinal rheumatism may occur in the first period of the disease, but I have noted it more frequently, in a severe form at any rate, following one or more "relapses" in the form of tonsillitis. Next, in some children we have quite severe tonsillitis, with considerable swelling of the throat and glands, involvement of the nasal passages, and frequently otitis. Lastly, we have the sudden development of a local infection, passing into a chronic rhinitis, adenitis and tonsillitis, persisting over many weeks, with slight fever or none at all, and occasional minor exacerbations.

All these later conditions are very common in children under five, fairly common in children from five to ten, and uncommon in older children and adults.

I am inclined to think that nephritis, what we may call a "toxic" relapse, is to be explained most often on the first hypothesis. I am bound to admit that I first formed this opinion on insufficient evidence. I saw nephritis developing while patients were still in bed and to a certain extent isolated from other sources of infection, while on the other hand the more "septic" processes usually began after the children were up and in closer contact with others. But cross-infection was not excluded as a source of the nephritic relapse. Lately, however, I have watched the occurrence of nephritis in more than one patient who has been nursed in a separate room, and in whose case the possibility of cross-infection has been virtually eliminated. We may therefore conclude that in some cases at any rate the development of nephritis can be fully explained by the first hypothesis. If a satisfactory and reliable interpretation could be placed on Dick test findings, it is here that by a series of such tests much valuable information could be gained. Unfortunately this is not so. There is still further evidence to bear upon the point, however, which can wait for the present.

The minor local reinfections of the throat, nose and ear and the lymph glands, and the subsequent sinking into a state of chronic or intermittent low grade infection, are no doubt very commonly due to the picking up of fresh strains of streptococci from other children. Closer contact, the common use of toys, and a less equable and uniform temperature than that experienced while in bed are all factors. Careful observations in various parts of the world have established the fact that in any group of children treated together the strains of hæmolytic streptococci in any one child are quite different in convalescence from those responsible for the primary disease, and are in fact interchanged. But again this explanation does not hold good for all cases. I have seen the typical reinfection in a child who has been isolated throughout his illness and presumably free from outside contamination.

This leads us, then, to ask, is there any third common factor? Not all children develop these relapses and reinfections or persistently carry infection for weeks and months on end. Not nearly all. And there is one factor which is of very great importance in the majority of cases, and the predominant one in many. I refer to preexisting unhealthy conditions of the nose and throat, particularly of the tonsils. Quite apart from the fact that in practically all fatal and very severe cases of scarlatina evidence of such can be obtained and, quite apart from the fact that scarlatina is rare in children whose tonsils have been removed, and comparatively rare in children whose tonsils appear even reasonably healthy, when one sees a child with large, lumpy, pitted tonsils flaked with exudate, or soft, boggy, red masses exuding pus, and little, hard, lumpy glands in the neck, one has gloomy forebodings of trouble in late convalescence, which are more often than not realized.

In my own experience all these late manifestations, classical relapses, nephritis, streptococcal reinfections, and the chronic "carrier" state are much more common now than they were formerly. During the last five or six years they have been much more common than during the preceding fifteen years.

We come finally to the very mildest types of scarlatinal infection, in which the usual clinical features of the onset may be slight or almost absent. Frequently there is no exanthem, sometimes no angina, and occasionally no noticeable fever. These cases are not often diagnosed, unless it be later, when the development of nephritis, otitis or adenitis brings the patient under medical attention. The discovery of desquamation on the hands and feet, or the history of the mild illness some two or three weeks before, makes the position clear. During the first ten years of my experience of scarlatina, nearly all the examples of nephritis were of this nature and came under notice in this way.

But this type of scarlatina does occur very frequently under conditions which should excite suspicion. If, with the development of definite scarlatina in a household, one finds immediately preceding or following it, otitis or tonsillitis in another child, the scarlatinal nature of the latter should be suspected. If, then, cultures of swabs from the tonsils or ear discharge show hæmolytic streptococci, the illness should be regarded as scarlatinal. Whatever doubt there may be as to the significance of the presence of hæmolytic streptococci in what we may call normal throats, there can be no reasonable doubt in the circumstances mentioned.

Now all forms of the primary disease coming under notice today were common forms sixty or seventy years ago. The fulminating toxic and septic varieties, the so-called "typhoid" form, which is most commonly fatal nowadays, ending in general sepsis, pyæmia or bronchopneumonia, the moderate illness showing the ordinary "text book" symptoms, the typical mild scarlatina, and, lastly, *scarlatina sine exanthemate*, were all well recognized and fully described, the only difference being that the severer forms were very much more frequent, as stated. But now we come to the really astonishing thing. I can find no record, not even a suggestion, of the existence in those days of what is now to my mind one of the most striking features of scarlatina. I refer to the development, from the third or fourth week onwards, of the state of chronic infection, with persistent discharges, and punctuated by minor inflammatory processes, to which insistent reference has already been made. The clinical appearance of children thus affected presents such an obvious picture, and the condition is so exceedingly common that the matter cannot be left where it is.

There are, of course, many divergences from the main type; many children are pale and pasty, others are fat and rather gross and flabby, others look fairly well and appear to keep in fair general health. The common features are hard, lumpy

glands, or sometimes masses of glands matted together on one or both sides of the neck, irritating and persistent rhinorrhœa with excoriation of the nares, inflamed fauces and pharynx, the tonsils pitted and dirty, often much enlarged, recurrent sores about the face, a tendency to infections of a suppurative nature round the finger and toe-nails, and often otorrhœa. From time to time exacerbations occur, coinciding with a fresh paronychia, a fresh tonsillitis, or an increase in size of the cervical glands with the development of pain and tenderness and generally accompanied by a rise in temperature for a few days. The whole condition may go on for months.

Now it is inconceivable that such a state of affairs could have been common in days gone by and escaped comment. The earliest direct reference I can find is that made by an Englishman, Millard, in the nineties. He stated his belief that some discharged patients "retained virulent germs in the skin, nasal mucus, otorrhœal discharges *et cetera*, despite all attempts to protect the outside world." Good heavens, "nasal mucus"! Not even here is a suggestion of the horrible rhinorrhœas which would attract the attention of a layman fifty feet away.

The rhinorrhœas of late convalescence are simply ignored in the literature of seventy years ago. Right up to the nineties the leading authorities considered that the patient was most dangerous to those about him in the early stages. Von Jurgensen, discussing the matter, wrote:

> . . . when the patient ceases to be able to transmit the disease. If we count in the exfoliation of the epidermis from the palms and the soles (some consider this unnecessary), the termination is long enough postponed. Such cases as these are probably, however, very rare.

Not a word about rhinorrhœa or otorrhœa.

A servant girl was detained in the Reichshospital in Christiana a month after the end of the fever, yet, on her return, and in spite of the fact that her clothing was disinfected, she carried the infection to the children in the house where she served. The official report on her case stated:

> It appears, therefore, as if the infectious substance had adhered to her person.

Von Jurgensen's comment on this is:

> If there is really no other interpretation possible, then the scarlatinal poison must be a still more wonderful entity than it is already considered to be.

The only conclusion I find it possible to form is that in those days it was unusual for patients to remain infectious for long periods. Further, that the spectacle which is now constantly before our eyes, of children in the chronic state of infection already described, was a rare one, so rare that it escaped detection. It is simply inconceivable that, if it had been otherwise, men like Murchison, Forster, Johanessen, Trousseau, Thomas and dozens like them, men who examined the disease as they saw it, to the smallest detail, and who knew far more about its symptoms and appearances than we do, would have missed it. To put it bluntly, the common condition of chronic streptococcal infection in the later stages of scarlatina, as we see it today, practically did not exist.

Is any explanation possible? I think that we can make an attempt to explain it in part. It is likely that a very large proportion of the children who now drift into the chronic stage, in days gone by never would have had the chance. They would have died. Diphtheria must have been responsible for an enormous number of deaths in children of this type, measles also, diarrhœas and dysenteries, and so on. But more than all, scarlatina itself would have killed them early. The terrible mortality of the toxic and "typhoid" types indicates the drastic weeding out of the weaker children. Of those who survived, a goodly proportion were healthy children, and as with healthy children of our own day, most of these recovered without complications or persistent discharges. Residual otorrhœas there were, we know, but if these did give rise to infection in others, the occurrence was ascribed to the house, the bedding, or some other inanimate object.

Is this a full and satisfactory explanation? It is not, and I do not think that we can formulate one. But I do think we can put into words a hazy idea that with the decreasing toxicity of the streptococcus there has grown up a mutual tolerance between us and it. When children two or three weeks after the primary stimulus of the initial attack receive a secondary stimulus in the form of a minor relapse, the toxins do not as a rule worry them very much. They are able to manufacture and, by the aid of successive stimuli, to keep up a supply of antibodies sufficient to protect themselves from gross toxic effects. Thus they sink into the state of the chronic carrier. Minor extensions of bacterial energies occur, involving the ears, the accessory sinuses of the nose and so on, and now and again the development of a common cold will light up those energies afresh. It is a depressing spectacle, but we must leave it at that.

What morals, if any, are to be drawn? I hesitate to say. One thing is certain, however: strong attempts should be made to protect small children from infection in convalescence by fresh strains of cocci. Isolation of patients in separate cubicles will fill the bill while they are confined to bed, but it is inhuman to condemn well convalescents to solitary confinement. The grouping of children in pairs, two to each small room, is better, but expense is a great objection. If medical supervision in proper home conditions is available, the early discharge and subsequent treatment and isolation at home of a large proportion of patients is to be seriously considered. I look forward to the day when most adults and older children, and not a few young children, may be discharged from hospital within the first fortnight. I would suggest that where there is a prospect of this being done, serum administration should be omitted in the early stage of illness and a small amount administered intramuscularly towards the end of the second week. In this respect we have done a little preliminary experimental work at Fairfield. Also I look forward

to the establishment of tonsillectomy in selected cases, at the earliest possible opportunity, possibly in the second week of the disease, as a definite line of treatment in scarlatina. The adoption of such surgical measures at such times as the eighth or ninth week, after chronic and extensive infection is well established, is much better than nothing, and excellent results are obtained in a fair number of patients, but in many they are rather disappointing.

The other moral to be drawn is that while we may hope, we can feel no security, no certainty, on the evidence before us, that the present mild type of the disease will continue indefinitely or that it will fade away and disappear. It may be that we and other Anglo-Saxon nations are gradually acquiring a general immunity and may ultimately arrive at the happy state of the Eskimos, the Malays and the Annamites. We have history to guide us, however, and to warn us, and may conclude this somewhat pessimistic address in the words of one of the old writers: "He is a wise man who remembers the treacherous nature of scarlatina."

INSECTS IN THEIR RELATIONSHIP TO INJURY AND DISEASE IN MAN IN AUSTRALIA.[1]

SERIES III.

By J. BURTON CLELAND, M.D.,
Marks Professor of Pathology, University of Adelaide.

To the Australasian Medical Congress held in Sydney in September, 1911, the first paper of this series was contributed, and this was followed by a second paper[(1)] submitted to the Congress held at Brisbane in 1920. Since the latter date, a number of additional records have been collected and are embodied in this, the third of the series.

Diptera.

Blowfly Maggots.—Dr. C. Sutton has informed me of an infection of a *primipara* in Carlton, Melbourne, in the summer of 1908.

She had a normal confinement and was nursed by her mother, who was clean and careful. The weather was very hot and the patient often lay with only a sheet on. The binder became disarranged. During the seventh day there was a rise of temperature and constitutional symptoms. The next day maggots were found whilst the patient was being cleaned.

I have been informed of two full-blood aborigines in the north of Eyre's Peninsula, South Australia, whose wounds became fly-blown. One had a fractured leg, evidently compound, as maggots entered the wound; the man died. In the other case fifteen large maggots were taken from a burn near the big toe.

Blowflies and the Finding of Murdered Bodies.—In April, 1926, two police officers at Boulder, Western Australia, disappeared, and it was believed that they had met with foul play. A search was made, and according to one of the newspaper references, the bodies were eventually found through a number of blowflies being noticed at the entrance to an abandoned mining shaft. As it appeared that these insects had been responsible almost entirely for the discovery of the victims of an atrocious murder, I made inquiries from Dr. Everitt Atkinson, Commissioner of Public Health, Perth, as to whether the blowflies were the first indication of the presence of the bodies, or whether a smell had been detected previously. The Commissioner of Police of Western Australia replied to my inquiry as follows:

The two police officers were no doubt murdered on April 27, 1926, seventeen miles south of Boulder City, and after some days missing a search was instituted, and on the evening of May 11 a report was received from a resident who had been out motoring about abandoned shafts, that he had detected a bad smell from a shaft almost five miles west of Kalgoorlie. Upon approaching this shaft blowflies were noticed at its entrance. They were few in number. Next morning a search was made, and the mutilated bodies and other articles were found at the bottom of this shaft, which was about sixty feet deep. The smell was first noticed and then the blowflies were seen.

Simulidæ (Sand-flies).—True sand-flies (Simulidæ) are comparatively rare in Australia. F. H. Taylor[(2)] mentions that only two species of *Simulium* had been hitherto described, *viz.*: *Simulium furiosum* Skuse and *Simulium victoriæ* Rouband. He describes a new species, *Simulium bancrofti*, from Eidsvold, Queensland, and refers to *Simulium furiosum* Skuse, from Narromine in New South Wales. I have met with a species of *Simulium* in rocky country at Bumberry in western New South Wales. The insects apparently followed the wallabies, but would leave them to attack men or dogs. *Simulium* larvæ have been found in streams at the Canoblas, near Orange. Mr. Taylor, in the same article, refers to *Culicoides townsvillensis* Taylor (Family Chironomidæ), a new species, as biting in the morning at Townsville, and he also describes as new *Culicoides multimaculatus*, from Portsea, Victoria.

Hume and Hovell,[(3)] during their overland journey to Port Phillip in 1824-1825, were tormented on the journey by swarms of little flies, which in all probability were a species of *Simulium*.

On November 4, 1824, Hovell wrote near Tumut (page 322) that they were tormented by day by swarms of little flies (not larger than a flea), but wherever they alight they draw blood, and by large mosquitoes at night, in addition to the blowfly. These "devils blow the blankets or anything else they light on."

November 7 (page 325): "We are again tormented by our new but constant companions, the sand-fly, and the mosquitoes; there is no getting rid of them, as they will stand fire and smoke as well as ourselves. My legs at this moment (a little above the socks) are one complete sore to within a few inches of the knee." On November 11 (page 327) Hovell says it was impossible to describe the torments from which they suffered. Next day (page 328) they were much tormented by the "devil-fly" ("I give them that name because they have a power to torment us and they are at the same time almost invisible"). On November 23 he wrote (page 340) that whenever they came near the mountains they were plagued by the devil-fly and mosquitoes, and on November 30 (page 348) he repeats this statement as applicable near the Goulburn River. On December 7 (page 354) the devil-fly and mosquitoes are mentioned, and on the twelfth (page 361), near Sunday Creek, they are again troubled by both.

[1] Read at a meeting of the South Australian Branch of the British Medical Association on July 30, 1931.

Hymenoptera.

Death from Bee Stings.—Dr. W. A. Hunter, of Salisbury, South Australia, has kindly given me the following details about W.S., an imbecile, an inmate of the Angas Home (but not deaf or dumb), aged twenty-four years.

This patient was found by the superintendent with his head and face covered with bees (a stray swarm) early on Friday, October 29, 1926. He brushed the bees off and a nurse applied a blue-bag. Dr. Hunter was called at once and found the patient's head, face and neck, including the eyelids and lips, thickly studded with bee stings. Dr. Hunter adds: "I have never seen anything like it. To pass your hand over his face was like feeling a chin with about three days' stubble on it. We applied ammonia and removed the stings. The face was greatly swollen and also part of the tongue, and he could not open his eyes. However, there was no œdema of the throat and the patient could swallow, though with difficulty, and was able to curse the bees pretty volubly. He did not seem to be in much pain. He vomited during the day, the vomit being very green and containing three bees. The next day he passed a number of bees *per rectum*. He lived until Monday midday, but died quite suddenly whilst talking to his mother. He had been conscious during the whole time, had been able to take food and apparently was in very little pain, possibly due to his imbecility. I was not present at his death, but was there shortly afterwards, and would put down the cause as being due either (i) to shock similar to that occurring four or five days after severe operations, or (ii) to infection through the facial veins."

Anaphylactoid Reaction to the Stings of Bees.—Dr. Gilbert Brown has kindly given me the following notes.

The patient during the last twelve years had had four attacks of a reaction following bee stings, in three of which he had been under Dr. Brown's observation. Immediately he is stung by a bee there is a feeling of nausea, coldness and faintness. He has either sat down or fallen down and has usually lost consciousness. On one occasion Dr. Gilbert Brown saw him within a couple of minutes of the attack and found him lying down unconscious, whitish-grey in colour, with a cold and moist skin. The pulse and heart sounds were very feeble and respiration shallow. There was a small wheal from the sting, but not much reaction locally either then or later. He has responded to stimulants and has recovered his usual health within a couple of days. Though now seventy-six, the patient is a very wiry little man in good general health.

Bee Stings.—Mr. Neville Cayley informs me that bee stings in his case give rise to rapid swelling of the affected part and that the glands enlarge a few hours afterwards to the size of marbles and remain enlarged for three days at least.

In the case of my son, when aged eighteen, several bee stings, inflicted when he was attending to a hive, were followed an hour or so later by scattered urticarial patches on the trunk and limbs.

Hornets.—Mr. William McLennan,[4] an ornithologist, was attacked by little yellow hornets whilst climbing for a strepera's nest in Cape York Peninsula in 1921. He was badly stung about the head, hands and back.

Stings by Wasps.—Sir Thomas L. Mitchell, the Surveyor-General, was attacked during his first exploratory expedition by a wasp, having "passed near a tree on which their nest was suspended." A footnote describes the insect as *Abispa Australiana*. A. Musgrave[5] states that this species is the well known yellow and black "mud dauber" wasp, and is now known as *Abispa ephippium* Fabr. He adds that F. Smith[6] points out that the passage in Mitchell's "Expeditions into Eastern Australia," Volume I, page 104, can hardly refer to this species, as it appears to have been some gregarious wasp by which the travellers were attacked.

Ants.—Backhouse,[7] in his account of his visit to the Molong district in 1835, states:

We lately heard of a man falling in a state of helpless drunkenness on one of the large, flat, loose anthills that were common in the bush. When found, he was lifeless, the exasperated ants having eaten the interior of his nostrils and throat.

A. J. Campbell[8] records that Mr. McLennan, when bird collecting on Groote Eylandt, Northern Territory, received a vicious sting by an ant in the eye, which affected his sight.

J.B., an old man, aged seventy-five years, was brought to the St. George District Hospital, Kogarah, New South Wales, on the night of January 12, 1926.[1] He had been stung by soldier ants which were said to be "the soldier ants usually found in the bush." On admission the patient was semi-conscious, with a pulse rate of 100, its volume and tension being good. His face and hands were blood-stained. On washing away the blood there appeared small areas where the superficial layers of the skin were eroded. These areas were about one-eighth of an inch square and were scattered over part of one cheek and part of the dorsal surface of the hands and feet. There was no sign of any other injury. The next morning he was quite conscious. He had not passed urine, so his bladder was catheterized some hours after admission. Only 150 cubic centimetres (five ounces) were withdrawn, and on examination this contained a heavy cloud of albumin. Later in the day he became drowsy, then unconscious, and appeared to be in a state of uræmic coma. He died early on the morning of January 14.

It seems very doubtful whether the patient's death was in any way due to the bites or stings of the soldier ants, these being merely incidental on an unconscious patient.

I have been informed that on the Lachlan, on one of Tyson's stations about 1883, a drunken boundary rider climbed a tree, thinking he was an opossum, and fell down on to an ant heap (*Iridomyrmex*—building big low mounds). The end of the nose and upper lip and penis were eaten away by ants.

In March, 1929, an inquest was held at Maitland, New South Wales, on the dead body of a baby boy, R.M.S., aged six months, whose body, much decomposed and mutilated by ants, had been found in the bush at Thornton. The child had apparently been abandoned by its mother in the bush—she was "sure it was dying." When found, the body was smothered with ants. The Government Medical Officer found the body so far decomposed that examination was almost useless. Death was attributed to exposure, assisted by the fact that the body was eaten by ants.

Coleoptera.

Longicorn Beetle.—W. W. Froggatt[9] mentions that *Epithora punctata*, a longicorn beetle that walks over logs of spotted gum (*Eucalyptus punctata*), never attempts to fly, but when picked up turns round and bites with its powerful jaws. This habit is well known to the timber-getter.

Lepidoptera.

Bugong Moths and Vomiting.—Dr. George Bennett, in his "Wanderings in New South Wales,"

[1] I am indebted to Dr. K. T. Helms for the notes of this case.

described cakes made by the natives from the bodies of Bugong moths from Bugong Mountain. It appears[10] that on the first few occasions when the cakes were used by the natives, they made them very ill and subject to severe attacks of vomiting, but after a few days they began to thrive and fatten on them.

Hemiptera.

A. Musgrave,[11] when exhibiting specimens of Capsid bugs, *Calocoris norwegicus* Gmel. (*Calocoris bipunctatus* Fabr.) from West Devenport, Tasmania, mentioned that H. Stuart Dove, when sending them, stated that a young man who had been bitten by them, had a raised, suppurating swelling on the back of his hand that took ten days to heal. Possibly here an accidental secondary infection accentuated the ill-effects.

T. G. Campbell[12] states that the sting from *Enithares bergrothi*, a back-swimmer, is said to be somewhat similar in its effect to that of a bee, the wound being accompanied by swelling.

One of the earliest references to the occurrence of bed-bugs in Australia is in the account of a journey from Sydney to Bathurst in 1822, contributed by Mrs. E. Hawkins.[13] They were found at night crawling by hundreds in a hut near Springwood in the Blue Mountains.

W. A. Goodacre,[14] apiary inspector, mentions that assassin bugs of several species lurk in flowers and sting bees on which, with other insects, they feed. On being handled they may sting or bite severely.

T. Steel[15] reports that in Sydney he felt a sharp puncture on the back of his hand and found a Rutherglen bug (*Nysius vinitor* Berg.) apparently sucking blood. He also mentions a variegated plant bug which inflicted a sharp painful puncture.

Lice.

The following quaint legend of the blacks is taken from Backhouse's[16] "A Narrative of a Visit to the Australian Colonies, 1843." It would appear from this account that lice must have affected the blacks before the arrival of Europeans.

Kurrurkurran is a place at the north-west extremity of Lake Macquarie, where there is almost a forest of petrified wood. The tradition of the aborigines is that this was formerly one large rock, which fell from heaven and killed a number of blacks who were assembled where it descended, by the command of an immense guana, or lizard, that came down from heaven for the purpose, in consequence of his anger at their having killed lice by roasting them in the fire. Those who had only cracked them, are said to have been previously speared to death by a long reed from heaven.

R. G. Waddy,[17] of Sydney, describes a case of intense conjunctivitis in a woman of forty-five, which was found to be parasitic, due to *Phthirus pubis* L.

Orthoptera.

Dr. C. Sutton has told me that at Muttaburra, in central Queensland, large black cockroaches were responsible for gnawing raw the skin near the bases of the nails of several fingers in the case of a servant girl and also of a billiard marker, both of whom had been sleeping under mosquito nets.

Mrs. Brenn,[18] of Broken Hill, when catching the long-horned tree "locusts" (Gryllacrids), which are really carnivorous crickets, has been savagely attacked and blood has even been drawn.

General.

A. Musgrave[19] has an article entitled "Some Australian Insects Injurious to Man." He refers to cockroaches nibbling the toes of would-be sleepers. Amongst the long-horned grasshoppers (Family Phasgonuridæ) he mentions *Anostoma australasiæ* and *Anostoma erinaceus* as having the head and jaws strongly developed and biting, the latter viciously. The ground beetle (*Trogodendron fasciculatum*, Family Carabidæ) bites savagely when captured, and holds on so tenaciously that its body may be pulled off its head. The golden spotted ground-bug (*Pirates ephippiger*), found round the Myall Lakes, is able to drive its stiletto beak into the hand of the person interfering with it.

Amongst those insects aggressive by stinging, he mentions the sand-wasp (*Exerius latertus*) as capable of causing intense pain, and that several stings from one of these insects may be attended by serious results. Paper-nest wasps (*Polistes*) may also cause annoyance. He mentions the bull-dog, soldier and green-head ants as stinging. The meat or mound ant (*Iridomyrmex detectus*), so abundant throughout Australia and constructing the large, curious nests so frequently met with, can bite savagely, but does not sting.

He also refers to mosquitoes, biting flies and fleas.

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A STUDY OF BACTERIOPHAGE IN RELATION TO INFANTILE BACILLARY DYSENTERY.

By F. M. BURNET, MARGOT McKIE and I. JEFFREYS WOOD.
(From the Walter and Eliza Hall Institute and the Children's Hospital, Melbourne.)

DURING the two summers 1929-1930, 1930-1931, we have carried out a study of the possible part played by bacteriophage in recovery from bacillary dysentery in infants. During the first season ordinary methods of treatment only were used and an attempt was made to correlate the spontaneous appearance of phage in the fæces with the clinical course and outcome. Last summer a highly active polyvalent Flexner phage mixture was administered therapeutically to about half the patients, an attempt being made to have comparable series of control and treated cases.

Infantile dysentery in Melbourne is predominantly due to Flexner type bacilli, our own and most of the previous investigations (Burnet, McKie and Wood, 1930, Webster and Williams, 1925) showing a predominance of the *W* type of Andrewes and Inman (1919). All the other types have been found in smaller numbers, and infections due to Sonne's bacillus are also not uncommon. The Shiga type is extremely rare in Australia and has not been found in any of the infants examined. Like most other investigators, we have encountered a number of strains resembling Flexner bacilli in their general characters, but not corresponding serologically to the recognized races. Elsewhere we have provisionally described two groups of such bacilli as races *T* and *U* of *Bacterium flexneri* (McKie and Burnet, 1931).

The Spontaneous Appearance of Bacteriophage in Infantile Dysentery.

The method adopted was to choose from a freshly passed stool a few loopfuls of muco-pus and add this to a tube of nutrient broth (pH 8·0). Some of the material was immediately plated on McConkey's medium and the broth tube incubated overnight. Next day the culture was filtered through a Seitz disc and the filtrate stored in the refrigerator until the responsible strain of dysentery bacillus had been isolated and identified. The filtrates obtained at various stages of the illness were then tested on the homologous strain by adding successive dilutions of the filtrate to areas of an agar plate previously spread with a few drops of a broth culture. When lysis occurred, representative single plaques were "subcultured" and the type of phage present determined according to the method previously described.

The results of these investigations have been already published (Burnet, McKie and Wood, 1930). There was a suggestion that in two or three cases in which an active phage appeared early, recovery occurred more rapidly than the others, but there was no clear correlation between the activity of the phage present in the fæces and the clinical course. The one point which emerged clearly, was that spontaneous appearance of an active phage was much rarer in young infants under twelve months than in older infants. It seemed suggestive that in the season 1929-1930, of seven patients under nine months old at admission, only one showed the presence at any time of an active homologous phage, and this patient was also the only one to recover in the group. The same relative absence of spontaneous phage in young infants has been observed in last summer's cases. Pooling the results over the two years, the occurrence of spontaneous homologous phage in the two groups was as shown in Table I.

TABLE I.

Occurrence of Spontaneous Phage in Infantile Dysentery.

Group.	Highly Active.	Weak.	Nil.	Total.
Under nine months .	2	5	12	19
Over nine months .	19	24	20	63

Therapeutic Administration of Phage.

A highly polyvalent Flexner phage was prepared in which were included all the types of dysentery phage which we have described. It was grown at the expense of recently isolated bacilli of types *W*, *Z*, and *VZ*, the three most frequently encountered types. Lysis was rapid and complete, and unfiltered lysed cultures remained clear for over a week when kept at 37° C. The lysed cultures were filtered, ampouled and tested for sterility.

In the treated cases the routine was to administer two cubic centimetres by mouth in a little water as soon as a specimen of fæces for bacteriological examination had been obtained. A further cubic centimetre was given on each of two successive days. Examination of the fæces showed that this was sufficient to insure the presence of an active phage in the lower bowel.

Analysis of the results shows very little evidence that the administration of phage had any influence on the disease. If we omit cases due to Sonne strains and atypical Flexner types (race *T*), 58 cases of true Flexner infections are available, 25 treated, with 33 controls. In the treated series 11 deaths occurred (44%), in the controls nine (27%). Clinically, no definite good or bad effect could be ascribed to the use of phage, and the higher mortality in the treated series is in all probability fortuitous.

More detailed analysis shows that two factors, the age of the patient and the time elapsing between the onset of the disease and admission to hospital, are apparently of considerably more significance in determining the outcome than the administration of phage. Table II shows the results arranged in regard to these two factors.

It is clear from this table that any influence which the administration of phage may exert on the disease, whether beneficial or harmful, is overshadowed by the two other factors of age and the duration of the disease before hospital treatment is instituted. In order to determine finally whether phage has any significant effect, a much larger series would be necessary, so that groups of treated and untreated cases would be available, comparable not only in regard to these two factors, but also

TABLE II.[1]

Duration Before Admission.	Phage Treated Cases.					Control Cases.					Combined Totals and Mortality Percentages.	
	Months. 0-6	7-12	13-18	19+	Total.	Months. 0-6	7-12	13-18	19+	Total.		
0-1 day	2/0	2/0	—	2/0	6/0	0/2	1/0	2/1	5/0	8/3	14/3	21·5%
2-3 days	—	1/2	1/0	—	2/2	—	3/1	1/0	2/1	6/2	8/4	33·0%
4-7 days	0/2	0/2	2/4	2/0	4/8	0/3	3/0	3/0	2/0	8/3	12/11	48·0%
8-14 days	—	0/1	—	—	0/1	—	1/0	0/1	—	1/1	1/2	67·0%
15 + ...	—	—	1/0	1/0	2/0	—	—	1/0	—	1/0	3/0	—
Total	2/2	3/5	4/4	5/0	14/11	0/5	8/1	7/2	9/1	24/9	38/20	34·5%

[1] In this table the upper figure represents number of patients in the group who recovered, the lower the number who died.

in regard to type of Flexner bacillus. Our *VZ* cases showed an unduly high mortality, while in the atypical *T* group (not included in the figures) no deaths occurred amongst either treated or untreated patients. When one considers in addition that the mortality from the disease varies greatly from summer to summer, it seems very unlikely that any slight beneficial or harmful effect will ever be demonstrable.

We therefore feel justified in saying that neither statistically nor by clinical observation was any striking benefit or harm derived from the administration of phage, and further, that it seems quite impracticable to determine whether any slight benefit is obtainable in certain cases. The only encouraging feature of the present series was the recovery of two phage-treated infants under the age of six months, the only recoveries amongst nine infants of this age group. Taken along with the relative absence of spontaneous phage in young infants, this suggests that it may be worth continuing the investigation with such cases.

Discussion.

The negative results of this study are in line with most recent reports on the treatment of infantile or adult dysentery by bacteriophage (Davison 1922, Munter and Boenheim 1925, Fletcher and Kanagarayer 1927, Riding 1930, Taylor, Greval and Thant 1930), and it is very difficult to see why they differ so extraordinarily from the results reported by d'Herelle. The phage used complied with d'Herelle's criteria of activity—it included the commercial *"Bact-dysenterie-phage"* sponsored by d'Herelle — and was in addition very highly polyvalent.

An observation which we made on several occasions, may throw some light on the complete failure of bacteriophage to influence the disease. When a fragment of muco-pus from the stool of an infant who had received phage a day or two previously, was plated on McConkey's medium, in two or three instances a very characteristic picture resulted which demonstrated clearly the coexistence of phage and of sensitive Flexner bacilli in the stool. On the heavily inoculated part of the phage there were a few coliform colonies only, but where the material was highly diluted in the final strokes, numerous Flexner colonies, some "nibbled," but mostly of normal appearance, were visible. Subcultures from the Flexner colonies gave typical sensitive cultures which were completely lysed by the phage derived from the same specimen of fæces. It is clear that under the conditions existing in the inflamed bowel a highly active phage and normally sensitive bacteria can continue to coexist.

Bacteriophage lysis in its classical form seems to be essentially a test tube phenomenon requiring for its manifestation an actively growing substrate organism in a medium in which free diffusion is possible. In the body or in such *in vitro* experiments as those of Dressel and Lewis, who found that *Bacillus coli* and an homologous phage could coexist indefinitely in tissue cultures of fibroblasts, the two entities are present in an organized semi-solid colloidal matrix where neither free multiplication of the bacterium nor diffusion of phage can occur. Undoubtedly some increase of phage at the expense of pathogenic bacteria can occur in the body, but the bacteria destroyed are probably those in the bowel lumen rather than those actually producing the pathological effects in the tissues of the intestinal wall.

Conclusion.

Therapeutic administration of a highly active polyvalent Flexner bacteriophage has no influence on the course of infantile dysentery.

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BRILL'S DISEASE OR PSEUDO-TYPHUS.

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HISTORICAL old world typhus is a very well known and clear-cut disease with the following salient characters—sudden onset, mottled rash, high fever of two weeks' duration, and a positive Weil-Felix reaction given by the blood. It shows a preference for temperate or cold countries, is highly infectious, is louse-borne and generally a very fatal disease.

Chiefly in tropical and subtropical countries within comparatively recent years there has been recognized a disease which looks like typhus, but is not quite the classical disease—what one might call pseudo-typhus. It is a much milder disease, is not highly infectious, and is not louse-borne.

Brill, of New York, first published an account of this disease in 1897, and later, in 1910 and 1911, in the *American Medical Journal*, over 200 cases were quoted by him as having occurred in New York and other parts of America. After careful consideration of the whole symptom complex he came to the conclusion that the disease resembled typhus, but that it definitely was not typhus.

This disease or group of diseases has now been reported from many different countries, United States of America, Mexico, India, Malay States, Australia, East Africa, South Africa, West Africa, Marseilles and Rome.

It has now been extensively studied and the results from the experimental side have rather changed the views held about the disease from those held by an early writer like Brill; practically all writers on the disease now frankly regard it as true typhus just in a mild form. The result is that most investigators have now for a number of years been in quest of a virus and vector.

The experimental findings which caused this change of view, are shortly as follows.

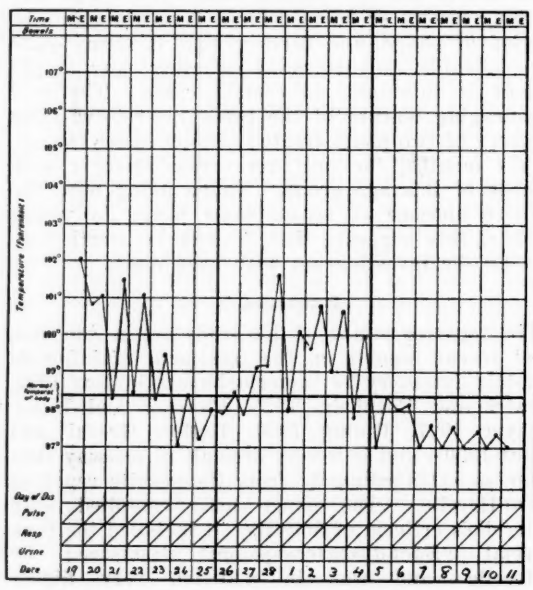
Anderson and Goldberger, whose work was confirmed later by others, found that there was a cross immunity between true typhus and Brill's disease, as shown by experiments on animals; monkeys inoculated with blood from Brill's disease patients were afterwards found to be resistant to inoculations with blood from typhus patients, and *vice versa*.

The serum of a patient with true typhus powerfully agglutinates a special strain of the proteus group of organisms, *Bacillus proteus* X, so does the serum of a Brill's disease patient (Weil-Felix reaction).

Maxcy found that the blood of a Brill's disease patient injected intraperitoneally into a male guinea-pig gave reddening of the testicles, similar to what we find in the case of the injection of typhus blood.

Summary of the Disease.

Brill's disease is a remittent fever lasting about fourteen days. The onset is usually sudden, accompanied often by severe headache, shivering and vomiting. The patient usually takes to bed. Headache is often intense, the temperature is usually 38·3° to 39·4° C. (101° to 103° F.) for the first week of the disease; during the second week it is a little higher (see accompanying chart). About the end of the first week the rash appears and the patient feels worse; this is usually the time poorer patients reach hospital. At this stage the face is red and swollen, the conjunctivæ congested, the eyes glistening, and the headache still severe. There is pronounced cough, but little sputum; what there is is very sticky, and generally the appearance is that of a very toxic condition. The high temperature continues with daily remissions of about one degree Fahrenheit till the twelfth day, when it falls by crisis or rapid lysis to normal by the fourteenth day. The pulse is slow compared with the rise in temperature. The rash is very characteristic of the disease; it usually begins towards



Temperature Chart of Patient with Brill's Disease.

the end of the first week and has disappeared before the temperature reaches normal. When fully developed, it shows a dark subcuticular mottling with red points, rather as if we took the mottling of measles and superimposed on it the red points of German measles. It mostly occurs on the chest, abdomen, back and limbs, and is often most marked on the back. In most well developed cases of the disease there is some delirium or mild mental wandering at nights. The patient towards the end of the disease appears very ill, but on the temperature falling, convalescence is very rapidly established. The mortality is usually quoted at 2%, but there have been higher rates, particularly as quoted in the Bulletin Number 1 of 1930 from the Institute for Medical Research, Federated Malay States, where a great deal of research work has been carried out on this disease for several years. When death occurs, it is usually due to lung complications, and on *post mortem* examination the most noticeable finding is fibrinous pleurisy with consolidation of portions of the lungs.

Probably the first cases of Brill's disease to be described in Australia occurred in Queensland in 1910, though I have not access to the descriptions, the authors being, according to the Federal Health Council Report, 1930, Smithson, Clark, Breinl, Fielding and Priestley. Later, 1921, numbers of cases appeared around Adelaide, and long and accurate reports were made by Hone in several papers, the diagnosis of Brill's disease being made then, and confirmed by Dr. Penfold, Melbourne, by the Weil-Felix reaction. Later, reports were published by Wheatland (1926), Holmes and Richardson (1930), D. A. A. Davis (1930) and others, and a very comprehensive review of the whole typhus series of diseases, with their scientific investigation up to date, by Adey, in the Federal Health Report noted above.

The first case to be recognized as such in Western Australia occurred in Perth (1926) and was diagnosed in the Perth Hospital. Two of us from the Health Department visited the first two groups of cases which occurred in connexion with food stores in Perth and Fremantle. We noted the possibility of rats being connected with the disease, and the blood from several was examined for the Weil-Felix reaction, but no positive result was found, nor have we found one since, nor have we ever had any knowledge of mortality among rats or mice in infected dwellings.

Our cases in Western Australia have occurred in Fremantle (coast port), Perth (twelve miles up the river from Fremantle), Geraldton (coast port, 200 to 300 miles north of Perth), and an odd case or two from inland towns.

Most of the cases occur from December to May (the hot season). During the rest of the year all we get is an odd case or two each month. Our total for the year has been 34, and my impression is that the disease is increasing in frequency. Since 1926 all specimens of blood for Widal test have been subjected to the Weil-Felix test as well. We have found no blood reacting to the Kingsbury strain of *Bacillus proteus* X.

I had an opportunity of seeing the first patient with the disease, referred to above, at the Perth Hospital. By that time his temperature had returned to normal and, of course, there was no rash remaining. He looked very exhausted, and on hearing the clinical history, the most arresting statement was that there had been great abdominal distension throughout the disease. In fact, so urgent was it that the rectal tube had to be used for a week, even after the temperature became normal.

Bacteriological Investigations.

After seeing numbers of other cases this persistent abdominal distension was seen to be quite unusual, but at the time it served to give me a theory on which to work, the idea being that there was a difference between typhus and Brill's disease, as shown by the different epidemiology, and that this might be due to the former being a virus disease and the latter a bacillary form of something similar, and that the abdominal symptoms might point to the bacillus having an intestinal habitat. Investigations were at once commenced and have been kept going during the last five years. Blood culture work and plating of fæces, the one or the other, or both if possible, were carried out in every case from which specimens were procurable. In doing blood culture extensively, in which sterility of technique is so essential, I expect we had, what other workers have had too, numbers of organisms of contamination. However, as I had resolved to do the work systematically, whatever was grown was treated seriously, and every organism, whatever its nature, was on its first appearance fully investigated as to its serology. First it was tried for agglutination by the serum of a Brill's disease patient and then it was injected into a rabbit with the hope of obtaining a serum which would agglutinate *Bacillus proteus* X19.

In the persistent hunt for a Weil-Felix reaction many scores of rabbits must have been used, and organisms employed ranged from cocci to yeasts and spore formers. During all this time my appreciation of the stability of a *Bacillus proteus* X emulsion rose very high; no serum from any of these numerous rabbits produced even a partial agglutination.

When I was engaged on this work it seemed that during this disease, as has been noted in true typhus, one is very liable to get accidental organisms swept into the circulation, having no causal connexion with the disease. For instance, we had one rather peculiar occurrence with one of our blood cultures. A sample of blood *T* was sent from Fremantle Hospital to the laboratory to be tested for the Weil-Felix reaction, the condition having been diagnosed clinically as Brill's disease. The serum gave a powerful agglutination of X19 up to one in 800. A sample of blood was requested

for blood culture, and, having been obtained and cultured, gave a profuse growth of *Bacillus fæcalis alkaligenes*. A third sample was then obtained in the expectation that it would agglutinate the organisms isolated from the blood, but all it could do was to give a ± one in 100 agglutination, while still giving a powerful agglutination of X19.

An agglutinating serum was then prepared by injecting one cubic centimetre of a dilute bacterial emulsion of the organism into a rabbit subcutaneously. The one injection was enough, as after five days the rabbit's serum strongly agglutinated its homologous organism up to one in 300.

One is unlikely to get an accidental contamination of culture medium by *Bacillus fæcalis alkaligenes*, so in all probability it was a bacteriæmia. From warm countries numbers of cases appear in the literature of *Bacillus fæcalis alkaligenes* bacteriæmia or septicæmia accompanied by fever, but usually they do not seem to give good agglutination against the organism, yet agglutination apparently is very easily obtained artificially. It would be interesting to test such cases in future by *Bacillus proteus* X19 to see if they would agglutinate that organism.

Seeing that no success had attended our blood and fæces examination, some other method of attacking the problem was considered.

When patients with Brill's disease die, they mostly die from pneumonic complication; hence it appeared that an examination of the sputum might give some information.

Cough is a constant symptom of this disease, but most of the patients do not seem to expectorate, especially those with the milder infections, sputum apparently being so scanty that it is swallowed. Sputum, however, can be obtained if the patient be encouraged to spit into a vessel and preserve it for examination purposes or if an expectorant mixture be administered.

One sample of sputum was obtained from Fremantle Hospital in September, 1930, and two more immediately afterwards from Perth Hospital. It was noticed that they were all of one type—white, glairy and very sticky. On looking up reports of the disease, there is only one reference I can find to sputum, namely, one by Dr. Davis, Brisbane (THE MEDICAL JOURNAL OF AUSTRALIA, June 7, 1930), in which he states that the sputum was sticky. All the samples I have had since maintain the same noticeable features of whiteness, glairiness and stickiness; some are almost like rubber in consistence. As Brill's disease has characters akin to the other acute exanthemata, it was resolved to concentrate first on the streptococci and other Gram-positive organisms, particularly on those showing viscidity. Numbers of different types of streptococci were picked off plates, also staphylococci and diphtheroids, the last of which are often very numerous. The organisms which grew in tough colonies were the object of special attention, but gradually a large number of those and also other organisms were injected into rabbits and the sera tested for their power of agglutinating *Bacillus proteus* X19.

No success having been obtained with the Gram-positive, attention was next turned to the Gram-negative organisms.

A sputum was obtained from Dr. Lucraft in May, 1931, and plated out. In two of the plates an odd colony or two of a Gram-negative organism was found, which was extremely sticky. On culturing on agar, it grew as a raised, thick, very viscid streak of pellicle. Single colonies have a central prominent point with lines radiating from it to the periphery. The organism itself is so protean, especially on different media, as to make description difficult. It varies from a stout oval bacillus, through diplobacilli with sharp pointed, not rounded ends, to long whip forms. It is not motile and does not ferment glucose, lactose, mannite, saccharose or dulcite, either in peptone water or broth. It does not form indol. It does not liquefy gelatine, but then it will hardly grow at all at room temperature. It prefers moist media. While being subcultured daily, it grows luxuriantly, but dies out with disconcerting rapidity, on two occasions having been almost lost through delay in subculturing. Since being isolated and having been subcultured so often, it has now lost a little of its mucoid character and now presents a rougher growth. It now has a proportion of rough variants. There has not been time to test the rough and smooth colonies separately, so the experiments described below have all been done with the mixed culture.

An agar slope was washed off with six to seven cubic centimetres of saline solution. Half a cubic centimetre of this was injected subcutaneously into a large rabbit (Belgian hare) followed five days later by 0·5 cubic centimetre intravenously. The antigen being very mucoid, it was feared it might not be quite suitable for intravenous injection. As a matter of fact, there was some embarrassment, as the rabbit did not feed for some hours afterwards, but next morning was very lively. Two cubic centimetres of blood were drawn off five days afterwards, and the serum gave a powerful agglutination with *Bacillus proteus* X19, up to one in 300, also, fortunate to relate, it gave the granular type of agglutination.

The *Bacillus proteus* X19 in use here is, I believe, number 67, National Collection of Type Cultures, obtained from the Commonwealth Serum Laboratories, Melbourne.

Another experiment was done to test whether the agglutination was due to the heat-stable antigen.

Two slopes were washed off as before. One was heated to 100° C. for half an hour, the other remaining unheated. In this case both injections of each antigen (0·5 cubic centimetre first dose and one cubic centimetre second dose) were injected subcutaneously only, in case the mucoid material injected directly into the blood stream should have a physical effect on the serum of the rabbits. The rabbits were bled five days after the second injections. The agglutinations were not so powerful on

this occasion, only one in 100, but these tubes were quite cleared. Both sera agglutinated *Bacillus proteus* X19.

Post Scriptum.

It is now a few weeks since the above was written, and the organism has settled down to a more regular and smoother growth. The bacilli are practically all stout Gram-negative bacilli with pointed ends. It still retains the characteristic of dying out very rapidly. A culture after forty-eight hours in the ice chest loses its opacity, becomes clear and glassy and is very difficult to revive. We find the best way to revive it, if possible, is to subculture it into broth and after incubation to plate the broth thickly on to a moist plate. Peptone water does not seem to be rich enough for good growth; the organisms multiply very sparsely, but the medium becomes very mucoid. The organism is not pathogenic for rabbits, mice or guinea-pigs. An intraperitoneal injection produced no redness of the testicles in a guinea-pig. The intravenous route produces the quickest and best agglutinins. The serum does not agglutinate typhoid, the parathyphoid organisms or an ordinary *Bacillus proteus vulgaris*. It agglutinates the strain Warsaw of *Bacillus proteus* X, but, as noted above, not the Kingsbury strain.

As to what the next of kin of this organism may be, it is difficult to say at present. By growth temperature or by fermentation it does not fit in very well with three of the most probable groups, namely, the Friedländer group, the vibrio group or the proteus group, but later it may do so serologically. The only one I have tested it against so far, was a rather atypical Friedländer organism, and there was no affinity by the complement fixation reactions.

With regard to this organism being the cause of Brill's disease, I think we may as well sum up the points in its favour.

A case is diagnosed clinically as Brill's disease and confirmed by the serum giving a strong Weil-Felix reaction. A sample of sputum is obtained. It is of the type to which we have become accustomed, namely, glairy and mucoid. An organism is with difficulty cultured from the sputum. The organism grows as a sticky film, and in liquid media makes the medium very mucoid. On injection into a rabbit it gives a serum which strongly agglutinates *Bacillus proteus* X19, the type of agglutination being the granular type.

The claims of this organism will be very much enhanced if it is found to give any cross immunity in animals against true typhus and in that happy event its importance as well. This will have to be done overseas, as we do not have true typhus here. The difficulty I foresee will be its transmission alive by post on account of its unfortunate habit of dying out.

THE TREATMENT OF MYOMA OF THE UTERUS.

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FIBROMYOMA situated in the upper part of the uterus, not palpable externally and not producing symptoms, need not be operated on, but should be watched for the development of malignant changes which, according to the statistics of Fletcher Shaw, occur in 9·5% of cases.

Myomectomy is a valuable procedure in women during the child-bearing period of life, who desire children and who fully realize that a recurrence of the condition may take place, necessitating another operation.

Multiple myomectomy is not justifiable if the uterus is left so damaged that it is incapable of functioning. Victor Bonney's methods have increased the range of the operation in this respect.(1)

It must be remembered that removal of a tumour by myomectomy will not stop hæmorrhage; this depends upon hypertrophied endometrium, therefore curettage must also be carried out.

It has been proved that malignant changes and associated malignant diseases not infrequently complicate fibromyomata; this, of course, is an argument against myomectomy, as is also the fact that myomectomy leaves untouched the diseased cervix present in the larger number of instances.

The death rate after myomectomy is higher than after hysterectomy.

Radium.

Radium should play only a very small part in the treatment of fibromyomata. Jellett and Tottenham (page 243) state that:

Radium may do more harm than good in a cachectic patient. The danger of increasing the anæmia should be borne in mind. Inflammatory conditions in the pelvis contraindicate radium owing to the risk of peritonitis ensuing. A latent infection may be lit up.

And again (page 207):

Radium brings about shrinkage of the ovaries and is just as objectionable as the old treatment of removing the ovaries and leaving the uterus.

Fulkerson, at page 461 of his book, states:

Radium produces obliterative endarteritis with resulting atrophy of endometrium, myometrium, tubes and ovaries. A nucleus may be overlooked which may subsequently develop. Cancer may be overlooked.

In the third edition of Veit's hand book we find it stated that:

The action on the ovaries produces atrophy of the uterus and the uterine vessels, which consequently affects the fibroids by merely secondarily affecting their nutrition.

Curtis (page 80) writes:

Anæmic cases are poor surgical risks. They are still poorer subjects for radium. Pelvic infection with anæmia makes a particularly dangerous combination; it is my surmise that carefully selected statistics on radium treatment in anæmic infected patients would reveal an appalling mortality.

The field of usefulness for radium in the treatment of fibroid tumours appears to be constantly narrower.

Everything within range of radium—uterus, ovaries, vessels, cellular tissues—undergoes atrophy. We must recognize, therefore, that non-operative treatment with radium is more radical in its effects than surgical removal; the latter eliminates only the diseased tissues, leaving all else intact. Radium in a dosage sufficient to effect a cure is always destructive of delicate ovarian tissues.

These extracts from the most modern and most authoritative gynæcological text books, the authors of which are all experienced users of radium, should convince any unprejudiced surgeon that "radium should play a very small part in the treatment of myomata."

When one considers the difficulty of being certain that we have a fibroid only to deal with, and the still greater difficulty of being certain that none of the conditions which contraindicate radium, is present, we must admit that instead of radium treatment being devoid of mortality, it has actually a higher mortality rate than surgery. Although it may stop the hæmorrhage, it leaves the patient

uncured of her tumour, still liable to malignant or other change in that tumour and in the uterus, and with the impaired health which the frequently present septic focus in the cervix invariably brings about.

At the annual meeting of the Royal Australasian College of Surgeons we had cases presented to us which exemplified the truth of the foregoing in a very striking manner: (i) Radium had been applied for a supposed fibroid which subsequent operation proved to be a malignant tumour of the ovary. (ii) Radium had been applied for supposed fibroid which operation six months afterwards proved to be a malignant growth of the body of the uterus. (iii) Radium had been applied for a supposed fibroid of the uterus which operation disclosed to be a bilateral pyosalpinx.

Hysterectomy.

Subtotal hysterectomy has one merit, namely, to the novice it is easier; but, if the tenets of the College of Surgeons be adhered to—that no surgeon will undertake operations unless and until he has trained himself by assisting experts—total hysterectomy will prove equally easy and in every other respect far superior; the cervix, so generally lacerated, degenerated, infected and therefore a source of ill health, is removed, and the patient is rendered secure against the real risk of cancer development in the retained cervix.

By my method of total hysterectomy, described and illustrated in the *American Journal of Obstetrics and Gynecology*, 1917, and which I demonstrated a few weeks ago at the opening of the new wing of the Sydney Hospital, the hæmorrhage is slight because the cut is made inside the vessels supplying the cervix. It is also inside the attachments of the utero-sacral ligaments and endopelvic fascia. The keystone of the vaginal arch is not weakened. Subsequent examination discloses no vaginal shortening, and the firm rim of the cervical musculature can be plainly felt. All glandular epithelium has been removed. There is no longer any danger of cancer development. Convalescence is as uneventful as after subtotal hysterectomy. The mortality rate (514 consecutive operations with three deaths) is as low or lower than that shown in any published statistics of subtotal hysterectomy, and certainly lower than the mortality rate of radium treatment, when the unavoidable mistakes in diagnosis accompanying this treatment are taken into consideration.

Total hysterectomy can be advantageously employed when, during an operation for pelvic suppuration, it is considered desirable to remove the uterus and drain through the open vagina. The procedure was carried out in 18 of 93 such operations in the Sydney Hospital, which I reported in THE MEDICAL JOURNAL OF AUSTRALIA of November 6, 1920. All recovered.

Reference.

(1) V. Bonney: "The Technique and Results of Myomectomy," *The Lancet*, January 24, 1931, page 171.

Reports of Cases.

ENFORCED DELAYED TREATMENT OF ACUTE APPENDICITIS.

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R.S., A BOY of sixteen, with a past history of chronic nephritis following plumbism, was admitted on June 20, 1931, complaining of severe headache for seven days, with particular pain behind the right ear and radiating down the right side of the neck. His pallor was pronounced, œdema was not evident, the urinary output was diminished. A cloud of albumin was present in the urine. The systolic blood pressure reading was 225 and the diastolic pressure 170 millimetres of mercury. Examination of the fundi revealed albuminuric retinitis of both eyes.

Following admission he was very restless, with copious vomiting of greenish fluid.

Pathological findings were: (*a*) Blood urea, 100 milligrammes per 100 cubic centimetres of blood; (*b*) urea concentration test: 1·5% before urea was given, 1·55% two hours after urea was given; (*c*) urine contained red cells, pus cells, motile bacilli, but no casts.

Next day a "lagging lid" was noted when he closed the right eye, and in three days a well-developed facial paresis of Bell's type had appeared on the right side. It was demonstrated that the sense of taste was definitely affected on the right side of the anterior two-thirds of the tongue.

For seven days following admission he presented the condition of incipient uræmia. His mentality was dulled, he vomited frequently and his headache was constantly present. Treatment was the usual purgation and supply of bland fluids.

By the tenth day his general condition had much improved. He was taking sufficient nourishment, his bowels were acting freely and he was voiding large amounts of urine. On the fourteenth day he complained of generalized abdominal pain with tenderness, but without rigidity. He obtained relief by lying in a flexed position on his right side. Pressure on the abdomen seemed to relieve the pain. He vomited occasionally, but otherwise was quiet and almost lethargic in manner. Examination *per rectum* revealed nothing definite.

In view of the history of plumbism and the indefinite nature of the pain, a provisional diagnosis of lead colic was made. Within thirty-six hours, however, the pain was definitely right-sided, and a second rectal examination revealed a doughy, tender mass in the right lower quadrant.

His blood urea was now 85 milligrammes per 100 cubic centimetres of blood, and, of course, the urea concentrating power was as poor as previously. His general condition was so desperate that operative measures, even under local anæsthesia, were not considered advisable, and expectant treatment was adopted.

During the next seven days his abdominal pain was very acute and his treatment with morphine liberal. The temperature was of the swinging variety, but the pulse remained rapid, and presently a tender mass was palpable in the right iliac fossa. It was decided to confine the bowels, but turpentine enemata gave such relief that these were given daily for several days. The mass in the right side increased in size and tenderness to a point where resolution apparently took place, with a general regression of signs and symptoms. His progress through this condition was traced for twenty-one days.

Following his recovery, a further blood urea examination showed 85 milligrammes of urea per 100 cubic centimetres of blood. His diastolic pressure had, however, fallen considerably and the blood pressure readings were now 180 to 170 millimetres systolic and 105 millimetres diastolic, as compared with 225 systolic and 170 diastolic

on admission. The urinary output was good, with a cloud of albumin constantly present.

The facial paresis meanwhile had shown considerable improvement, and after seven weeks he was discharged from hospital in a much relieved condition.

Acknowledgement.

I am indebted to Dr. Ellis Murphy for permission to report this case.

Reviews.

REFLECTIONS ON LIFE AND LITERATURE.

WALTER MURDOCH, book lover and observer of men and manners, is one of those dangerous people with whom it is very difficult to disagree. His writing has a persuasive charm that is nowhere better shown than in his latest collection of essays entitled "Saturday Mornings."[1] He tells us that an essayist must be neither moralist nor pedagogue, nor yet a mere *farceur*; he must not rant nor rail nor "hold forth"; he must be informal but dignified, personal but unprejudiced; he must talk with ease and vivacity. It would seem then that essayists are born and not made. However it be, Murdoch adheres admirably to his own rules, and this book of essays is the best thing of its kind yet produced in Australia. Such breadth of outlook and personal charm, coupled with the light and illuminating touch of an experienced writer, cannot fail to claim interest. The essays have a decided literary flavour, and will therefore be most keenly appreciated by "bookish" people.

In "Saturday Mornings" we meet with books and people, manners and travels, then books again; the author's style is discursive, but never long-winded, and we usually find ourselves agreeing with his judgements. Under the formidable title "Sesquipedalianism" some sound advice is given on the art of writing. Having proved by demonstration that he is able to out-Johnson Johnson in the use of words whose length inspires terror and respect, Murdoch thus pithily advocates simplicity in writing: "Down with the abstract noun and death to the polysyllable." Dr. Johnson, in his opinion, was the world's worst writer of prose. Some most entertaining pages are devoted to criticism in literature. Murdoch asserts that there is too much second-hand appreciation about; that a "classic" is a book that everybody praises and nobody reads; and that true appreciation of books is a very rare thing. In spite of being self-styled "the meekest person in the world," in the essay "On Laughing at Tennyson" Murdoch sternly rebukes the type of modernism that holds up to scorn the immediate past, and in particular the Victorians. Modern criticism has for its chief weapon the "delicate, high-browed, gentlemanly sneer . . . the cheapest and the most ignoble of all forms of criticism." We must admit, however, to finding Strachey's method of historical biography vastly amusing, despite the warning that coming generations may consider us likewise ridiculous. It is with satisfaction that we read in the essay "On Nihilism in Literature" of the author's failure to appreciate the effusions of James Joyce and Gertrude Stein; it gives renewed confidence to find our deficiency shared by one whose opinions are so worthy of respect. To the would-be nihilist who objects that Shelley too, in his time, was thought a madman, we might reply that Shelley at least spoke English. In "Gentlemen of Fortune" fairies are dismissed as uninteresting and over-rated folk whose charm in no way approaches that of the pirate with his romantic magnificence, his bloodthirstiness and his descent from reality. Yes, a pirate is a fine fellow, but we confess to having a weakness for fairies and refuse to see them thus indignly banished.

But it is unnecessary to go further. Professor Murdoch is a delightful companion and a man after the book-lover's own heart. The scope of his reading arouses admiration and we envy the diversity of his literary taste as well as his vast capacity for enjoyment. These essays would certainly stimulate us to read widely if such stimulus were necessary; for the joy and infinite satisfaction that books can give are constantly revealed. The book lover has a kingdom of his own; a retreat from life's adversities and a precious weapon against the civilized curse of boredom. He may be regarded with good-natured contempt by the "man of action," but, as Anatole France tells us, *"c'est le cas de tous les amoureux."* And Murdoch wisely says that this love "makes young men old for their age and keeps old men young for their age."

[1] "Saturday Mornings," by Walter Murdoch; 1931. Australia: Angus and Robertson. Crown 8vo., pp. 240. Price: 6s. net.

PROGRESS IN PÆDIATRICS.

THE volume of the "Practical Medicine Series," 1930, devoted to pædiatrics fulfils a useful purpose in collecting in a small volume a summary of the main contributions to the study of disease of children during the year 1930.[1] The editor, Isaac A. Abt, in his introduction, lays stress on the fact that progress in pædiatrics in more recent times has been directed principally along the line of prevention, and he considers that the reduction in the infant mortality rate throughout the civilized world justifies the existence of pædiatrics as a specialty.

In the section of diseases of the newly born, the work of Goldbloom and Gottlieb in producing a condition in animals similar to *icterus neonatorum* by keeping them under reduced atmospheric pressure is described. No mention is made, however, of Hampson's recent work on physiological *icterus neonatorum* and familial *icterus gravis*.

The contributions to the subject of infant feeding are almost fantastic in their variety and unnaturalness. They evoke a protest from the editor, who writes: "This year's contribution to scientific infant feeding would be amusing if it were not so serious. The road to pædiatric fame seems to consist of inventing a new food mixture beautifully labelled, to be tested on the innocent babe."

The subject of sepsis in infancy and childhood is dealt with by Abt, who emphasizes the difficulties which the diagnosis of this condition in young infants may present. The control of diphtheria by prophylactic injections is discussed in several articles, as well as the antitoxin treatment of scarlet fever. In fact, the number of contributions in journals in various countries to the study of diseases in children is very large, and in this volume an excellent synopsis of many articles is given.

The book should prove useful to those interested in pædiatrics, because it gives abstracts of work done in various countries and reported in several languages, which is not easily obtained in other ways.

[1] "The Practical Medicine Series: Pediatrics"; Series 1930. Chicago: The Year Book Publishers. Crown 8vo., pp. 451. Price: $2.25 net.

THE BRAIN LAID BARE.

BERNARD HOLLANDER, who has just published a volume entitled "Brain, Mind, and the External Signs of Intelligence,"[2] is known as a write of semi-popular medical books mainly dealing with psychology and psychiatry. This latest contribution is likewise of a semi-popular nature and would, in our opinion, be satisfactory neither to the medical practitioner nor to the "man in the street," although packed with quotations from "the literature" and tastefully embellished with photographs of criminals and imbeciles, giving the semblance of a truly scientific tome.

The writer essays to prove a theory of cerebral localization, and with a wealth of clinical material and quotations from many a bygone journal, bolstered up with specious argument and uncritical assumption, claims to have proved that the "frontal lobes are destined for the intellectual

[2] "Brain, Mind, and the External Signs of Intelligence," by B. Hollander, M.D.; 1931. London: George Allen and Unwin. Demy 8vo., pp. 288, with illustrations. Price: 12s. 6d. net.

processes and the rest of the brain for the three primary emotions: the occipital lobes are for love; the parietal for fear; and the temporal lobes for anger. Their morbid manifestations are similarly defined: the frontal lobes for the early stages of mania and later for dementia; the parietal lobes for anxiety psychoses and melancholia; the temporal lobes for acute mania" *et cetera*.

The very essence of simplicity! Casanova was a great lover, therefore he had large occipital lobes. Dean Swift had middle ear disease, therefore he suffered from insane suspicion and irritability; Tennyson had a high forehead, therefore he was a genius. People with large heads show great mental capacity. People with small heads are often imbeciles! The hoarding and acquisitive instincts reside in the "upper anterior part of the temporal lobes." The food instinct (and here the author, on uncertain ground, quotes Paulesco) is to be located in the inferior part of the frontal lobe. "Epilepsy frequently involves the temporal lobes." But, as Dr. Hollander very well knows (but does not say), epilepsy just as frequently does not involve the temporal lobe; and all people with *otitis media* do not develop insane suspicions. Lobes do not make the lover any more than bumps make blockheads, knobs make nincompoops, or swelled heads make scientists.

To Bernard Hollander, M.D., the mind has no mystery and the brain is an open book, albeit a fairy tale. By diligently raking over the neurological, forensic and psychiatric archives of the last hundred years, he has amassed enough material to mislead and very little to guide. The problems of cerebral localization still retain their sphingine secret; and such an attempt as this to marry an adolescent neurology to a moribund phrenology can only result in what the Americans have so prettily termed "bunk."

NEUROLOGY.

PROFESSOR HERRICK makes a point of stating in his preface that his little work is, as its title implies, an introduction to neurology and nothing more.[1] Also in its progress through new editions the temptation to increase size has been sternly resisted. In substance it is an account of general principles bearing on the structure and functions of the nervous system. If, for example, the reader desires to refresh his knowledge on the constitution of neurones, the integrative action of reflexes, the functions of the cerebral cortex and so on, he will find all under the one cover. But since the book is of little more than pocket size, it must be obvious that it consists of a series of sketches in outline. The reader looking for details is referred to the many text books and atlases now available and of which lists are given.

The author is evidently a thoughtful and competent neurologist and one accustomed to teaching. The descriptive matter is accurate, notwithstanding the breadth and complexity of the subject. In all directions the research of others is adequately recognized, and in this way the publication is brought up to date.

THE DANCE OF DEATH.

WHEN Alfred Scott Warthin, a young medical student, was passing through Nuremberg in 1893 on his way to Vienna, he was attracted by a shop window containing a print of Dürer's "Ritter, Tod und Teufel" (Knight, Death and Devil). This awakened in him an interest in the representations of death in art. He started a collection and gathered pictures of the "Dance of Death." He has put his researches into an interesting volume: "The Physician of the Dance of Death."[2]

The dance of death is seen in wall paintings in different parts of Europe. In those paintings death is personified as a skeleton. Death takes representatives of each class of human society and dances with them to the grave. The pictures thus consist of representatives of each profession in the order of their importance, and each representative has a skeleton leading him by the hand. The author has not only studied the development of the dance of death, but has traced the position of the physician in these paintings. As time passes he advances in the social scale. The development of the dance of death may be divided into three stages: the pre-Holbein period, the Holbein period of the Renaissance, and the post-Holbein period, the period of Holbein's imitators. There is a chapter on the modern dance of death. The physician is generally to be seen carrying his urine flask; sometimes the physician carries the flask and sometimes the skeleton.

The author points out that in each new period of human thought the dance of death will express itself in new form corresponding to the predominant philosophy of the time. He states that an artist of the modern period is wanting to give adequate expression in some art form to the changed outlook of the twentieth century towards death.

The book is of interest from the historical, artistic and philosophical points of view and cannot fail to arouse enthusiasm.

Notes on Books, Current Journals and New Appliances.

THE STARS.

SIR JAMES JEANS, well known for his book "The Universe Around Us," if for no other reason, has written a delightful little book, "The Stars in Their Courses."[1] He has written an introduction to modern astronomy for those who have "no previous scientific knowledge of any kind." The subject matter of the book was first given as a series of wireless talks. The author's style is simple and his language non-technical. He takes his readers for a preliminary journey through space and time, and then he describes the sun's family. He shows how stars may be weighed and measured. He described the variety of the stars and then explains the wonder of the milky way. He goes into the depths of space and deals with the universe as a whole.

The book is fascinating in the extreme. We can well believe that people with any scientific training who read this book, will want to pursue the subject further and that they will tackle the author's more difficult works.

RICHARD BURTON.

THE story of Richard Burton has been told by Fairfax Downey.[2] Burton, known best perhaps as the translator of "The Thousand and One Nights," was a most remarkable man. His life was marked by a succession of adventures, any one of which far exceeds what the average person may expect to enjoy in a lifetime. It is said that he had Arab blood in his veins. Whether this is true or not, he certainly had an Arabian type of countenance; he could think as Orientals do and could live as an Arab without arousing suspicion. He visited the forbidden city of Mecca as a Mohammedan and endured hardships in so doing. The description of this journey alone makes this book worth while. His journeyings in many countries, his exploits with the sword, and his many-sided, virile character are described in a narrative that grips the attention.

[1] "An Introduction to Neurology," by C. J. Herrick; Fifth Edition; 1931. Philadelphia: W. B. Saunders Company; Melbourne: James Little. Post 8vo., pp. 417, with illustrations. Price: 18s. net.

[2] "The Physician of the Dance of Death: A Historical Study of the Evolution of the Dance of Death Muthus in Art," by A. S. Warthin, Ph.D., M.D., LL.D.; 1931. New York: Paul B. Hoeber. Double demy 9mo., pp. 158, with 92 illustrations.

[1] "The Stars in Their Courses," by Sir James Jeans, M.A., D.Sc., Sc.D., LL.D., F.R.S.; 1931. Cambridge: The University Press; Sydney: Moore's Book Shop. Post 8vo., pp. 198, with illustrations. Price: 8s. 6d. net.

[2] "Burton, Arabian Nights Adventurer," by F. Downey; 1931. Australia: Angus and Robertson. Demy 8vo., pp. 310, with illustrations. Price: 10s. net.

The Medical Journal of Australia

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Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

ENTERIC FEVER.

For several years the incidence of enteric fever in Australia has been becoming less. This fact has been noted in one departmental report after another, and various persons in responsible positions have doubtless rested more or less content in the knowledge that the glaring light of infection is somewhat dimmed, if indeed they have not claimed credit for the diminished intensity. To whom the credit for the lessened incidence is due does not really matter. The principles of preventive medicine and sanitary engineering have been applied to living conditions in certain areas, and a certain result has been achieved. The statistics are interesting. During 1930 the number of cases of enteric fever reported in the Commonwealth was 909; during 1929 the number was 949 and during 1928, 1,149. In 1921 the number of notifications was 2,849. All the States have shared in this reduction, and Tasmania has shown the greatest proportionate improvement since 1920. Queensland, South Australia and Western Australia experienced a slight setback in 1930. With upwards of nine hundred enteric fever infections occurring every year in the Commonwealth, we must conclude that an enteric fever problem still exists. To be content with improvement is easy; to hope for further amelioration without added effort is futile; to make the effort is an obvious duty.

The most important steps in the eradication of enteric fever from a community are the establishment of a good water supply and the installation of an adequate system of sewerage. If both these were available for all persons in Australia, the enteric fever problem would be much simpler. The population, however, is scattered and in many country districts water is scarce and a sewerage system out of the question. The problem, therefore, must be considered from two points of view—that of the cities and larger towns, and that of the sparsely populated areas. In most of the cities and large towns the water supply is adequate, but the same cannot be said of the means used for the disposal of human excreta. In many of the residential suburbs of some of the capital cities the objectionable pan system is still in use, and these suburbs are not of recent growth, for this might offer some excuse; they are long settled by purse-proud, self-regarding, superior persons who look rather disdainfully on their less pretentious but sewered brethren in other parts. It is only a year or two since a large portion of the city area in one capital was deprived of its pans. Unfortunately, when pans are tolerated, fly breeding is often encouraged in other ways—disposal of household refuse is not adequately controlled and scraps of food are offered as a sacrifice to flies at the roadside. Such statements as these would probably bring forth indignant denials from aldermanic gentlemen and shire councillors, but they are none the less true. In country districts where it is not possible to instal a sewerage system, the ideal to be sought is the use of either septic tanks or fly-proof closets. In South Australia septic tanks have been constructed in many places. The Central Board of Health has dealt with over 16,000 installations. Municipal councils and district councils in South Australia have the power to enforce the construction of tanks in towns or townships. In Queensland all closets in non-sewered areas are required to be fly-proof. These requirements are, of course, excellent, but it is to be noted that Queensland and South Australia are two of the States in

which the incidence of enteric fever was not reduced in 1930. Wherever provisos of this kind exist under State legislative enactments (and this refers to all States) they must be enforced. The conditions existing in some Australian towns are nauseating. It is not too much to suggest that medical practitioners resident in areas of this kind should become permanent conscientious objectors in this regard. Many of them, by reason of appointment as government medical officers or shire health officers, have special opportunities for putting forward their views.

Perfection of arrangements for sewage disposal are of fundamental importance, but given the best possible arrangements of this sort, there will be a residual amount of enteric fever which it will be difficult to eliminate. According to some investigators, two in every thousand persons are carriers of *Bacillus typhosus*. The discovery of a carrier may be exceedingly difficult. Readers are referred to two articles in this journal—one by Dr. W. J. Penfold, published about a year ago, and another by Dr. R. A. Southwood and others, published on October 31, 1931. The difficulties and rewards of perseverance are clearly shown by these authors. The diagnosis of mild or abortive infections may be difficult. Too much emphasis cannot be laid on the fact that enteric fever must be suspected in all cases of fever lasting a week or more without definite physical signs or a distinctive skin eruption; the application of the Widal test will probably settle the question. Further reduction in the incidence of enteric fever will be brought about on the one hand by vigilance of medical practitioners in diagnosis of obscure infections, in the search for carriers and in preaching the gospel of fly destruction; and on the other hand by the health departments in seeing that suitable regulations are framed and carried into effect, and in guiding the private practitioner in his search for carriers.

Current Comment.

UNEXPLAINED GASTRIC ANACIDITY.

THE terms achylia and achlorhydria in medicine are often confused. The former should denote absence of all gastric secretion (acid and pepsin). The latter term should signify absence of free hydrochloric acid, that is, gastric anacidity. To determine the acidity of the gastric juice two recent methods are available. Neutral red is excreted by the stomach and, when injected intramuscularly, its appearance in the stomach is parallel with the secretion of hydrochloric acid. In achlorhydria it does not appear at all. This is termed the chromoscopic method. In 1920 Popielski, by animal experiments, found histamine to be a powerful stimulant to hydrochloric acid secretion by the stomach. After the injection of five milligrammes of histamine, if the gastric mucosa be able to produce secretion, free hydrochloric acid will soon appear in the stomach. The chromoscopic test is the better, as neutral red is harmless, but histamine very toxic. Apparent achlorhydria may be due to neutralization of the acid, in which case the chromoscopic test will be helpful, but the histamine test may not. In true achlorhydria no acid is produced, but in false achlorhydria hydrochloric acid is produced, but it all becomes neutralized.

W. S. Polland and A. L. Bloomfield have contributed an article on unexplained gastric anacidity.[1] Introducing a small tube into the stomach after a fast of twelve hours or more, the fasting secretion was withdrawn and 0·1 milligramme of histamine per ten kilograms of body weight was given hypodermically. Afterwards the secretions were aspirated over successive ten-minute periods. Generally only a few mils of mucoid matter were obtained. In the opinion of Polland and Bloomfield, with the introduction of the histamine test, ideas of achlorhydria must be modified. Many persons failing to secrete free acid after a meal of bread, gruel or alcohol, give a normal response (or practically normal) after histamine stimulation. It would now appear uncommon for a stomach to be totally unable to secrete hydrochloric acid. This finding conflicts with earlier observations indicating that anacidity occurred in from 5% to 40% of people without apparent gastric disease. Polland and Bloomfield investigated the gastric secretion of about 500 persons, with and without digestive symptoms, by the histamine test. Failure to produce free hydrochloric acid (tested with dimethyl-amino-azobenzene) was noted invariably in pernicious anæmia, often in gastric carcinoma and occasionally in a group of patients having anæmia, stomatitis and diarrhœa and who might respond to liver therapy. Anacidity was also found in people with or without mild digestive symptoms, such condition being termed "unexplained anacidity." Of these cases 25 were noted by routine tests and were not expected from any symptoms. Every attempt was made to exclude other clinical explanations. Pernicious anæmia and gastric cancer did not enter into the diagnosis, and the patients were not seriously depleted by febrile, malignant or other grave disorder to justify the possibility of functional inhibition of secretion. In some cases

[1] *Archives of Internal Medicine*, September, 1931.

the same result followed repeated tests. None of the traditional causes of gastritis or damage of the gastric mucosa (spiced foods, alcoholic excess or other drugs) was conspicuous in the series.

Every writer has noted the latency of this condition; but mild indigestion and diarrhœa have been attributed to it. No characteristic manifestations were noted by C. S. Keefer and Bloomfield in one hundred cases tested by the Ewald meal; but in these cases it is possible that the use of histamine might have elicited free hydrochloric acid secretion. Of the twenty-five persons of the present series many were actually selected to serve in determining normal standards of gastric function. In these anacidity was totally unexpected. Only two patients were under forty years of age. The youngest was twenty-nine and the oldest seventy-five. Anacidity does not appear to be purely congenital. As regards sex, 72% were in males and 28% in females, but, as more tests were made on men, it is possible that there is no conspicuous difference in the sexes. When the information was available (twelve cases), there was no known family history of cancer, stomach disorder, anæmia or neurological disease. Occupations were miscellaneous. In only one case was there any suggestion of dietary inadequacy—a patient who had lived for three years mainly on cereals and toast. No factor of gastric irritation could be held responsible. Only two had been whisky drinkers, but in neither was the habit recent, and one other habitually took wine with meals. Only two had abnormal laxity of the bowels; ten were constipated and the others normal as regards bowel action. More than half never had indigestion or gastric symptoms. Three suffered from "gas"—a symptom so common as to be of no significance. Eight had definite gastric symptoms, including nausea, vomiting, indefinite abdominal pain or burning sensation in the stomach. It would be difficult to assume that anacidity *per se* produces definite symptoms, as only the minority manifested any, and these did not differ from those of patients having normal gastric secretion. In none was there a smooth tongue or palpable spleen suggestive of pernicious anæmia, nor were any changes detected suggestive of combined sclerosis. The Wassermann test gave no reaction in all, and only two presented anything unusual in the blood picture. X ray examination was made in twenty cases, nineteen showing nothing of importance. The other one displayed irregularity of the stomach quite unlike cancer. One of the patients was relieved by taking hydrochloric acid.

Polland and Bloomfield consider that, apart from pernicious anæmia and gastric cancer, some persons fail to secrete acid (or any actual gastric juice), and that such unexplained anacidities are met with in from 3% to 5% of patients in a medical clinic. The age incidence suggests an acquired rather than a congenital condition. It has yet to be shown what underlying lesion is responsible for the disorder of secretion and what the eventual effect will be on the general health. A further question arises, whether this form of achlorhydria is followed in any considerable percentage by gastric cancer.

Polland and Bloomfield make no mention of certain conditions in which hypochlorhydria or actual achlorhydria is considered to occur. These are rheumatoid (or atrophic) arthritis, thallium acetate poisoning and some cases of asthma in children. In chronic cholecystitis hydrochloric acid may seemingly be diminished or absent, but this may be due to an associated gastritis. In sprue the diminished or absent hydrochloric acid may be real or only apparent, a normal acidity being neutralized by the alkaline secretion of the duodenum. Further light on all these conditions might be gained by the systematic use of the chromoscopic and histamine tests. W. Zweig has asserted that true achylia may be associated with atrophic gastritis and carcinoma in which a gastritis occurs in addition to the toxic action of the cancer on the secreting mechanism. He also states that in some cases in which no histological change is found in the stomach, there is a constitutional or essential inability on the part of the stomach to produce a secretion, or the disability may be due to functional nervous disturbance. Knud Faber found no evidence of a constitutional form in young children, and considered the condition to be due to inflammation of the glandular parenchyma of the stomach. Erwin Kohn considers that achylia, however arising, tends to be associated with a blood picture which has a high colour index, like that of pernicious anæmia, but resembles it in no other respect. D. T. Davies considers that achlorhydria with secondary anæmia is a distinct clinical entity, responding to the continued administration of iron. Very much more research is required to elucidate the problems of this condition. If, after injection, neutral red be excreted into the stomach, that organ must be considered as able to produce acid, whether the acid be then found or not.

CARCINOMA OF THE THYREOID.

T. P. DUNHILL, whose work on goître has been followed with interest by surgeons in every part of the world, has made another report on carcinoma of the thyreoid.[1] His clinical conclusions should be emphasized. He states that a nodule in the thyreoid gland should not be regarded as of no importance and early changes in signs or symptoms should induce the medical attendant to investigate their cause. Histological examination should be undertaken whenever thyreoid tissue is removed, and sections should be taken from different areas. Advanced changes should not be regarded as hopeless. When the patient's condition justifies it, as much of the tumour as possible should be removed and X ray treatment started. This gives comfort to the patient and often prolongs life to a surprising extent.

[1] *The British Journal of Surgery*, July, 1931.

Abstracts from Current Medical Literature.

DERMATOLOGY.

Schizosaccharomycosis.

T. BENEDEK (*Archives of Dermatology and Syphilology*, October, 1931) states that *Schizosaccharomyces hominis* is a constant endoparasite of man, and that the dermatoses caused by it are merely "projections" of this endoparasitism on the outer surface of the integument. The author places in the category of schizosaccharomycosis the following conditions: seborrhœic eczema, *sycosis vulgaris*, seborrhœic rosacea, *pityriasis rosea*, Hutchinson's pompholyx, infantile eczema, *erythrodermia desquamativa*. By stained smears without cultivation *Schizosaccharomyces hominis* can be identified in its vegetative forms from vesicles and nodules. It can be distinguished from the products of the corresponding dermatoses by cultures of scales from the skin in an average mean total of 28·02% of the large series of its experiments, covering 1,560 cases. By this method it is possible to identify only such individuals of the parasite as have left the vascular system and, wandering through the derma and epidermis, have settled on the surface of the skin. By incubation of directly inoculated blood media cultures were obtained in 17·48% of 206 cases, the low percentage being due to the high inhibitive action that human total blood exerts on the growth. Through indirect blood cultures (the cantharadin method) the parasite was found in 60·5% of 279 cases. The degree to which the blood stream is flooded with *Schizosaccharomyces hominis* at a given moment can be shown only by centrifugation. By this procedure the parasite can be identified in the blood stream in its vegetative form and also when it has undergone phagocytosis by microphages and macrophages. In addition this procedure has made possible the identification of the parasite in the venous blood of the umbilical cord. While *Schizosaccharomyces hominis* occurred in pure culture in only certain of the cases in which scales were tested, and was there associated with pathogenic and non-pathogenic bacteria, in the blood stream it was always isolated and not associated with other organisms.

The Allergic Skin Reaction.

H. L. ALEXANDER (*Archives of Dermatology and Syphilology*, October, 1931) states that with the establishment of the identity between the localized œdema of allergy and the œdema produced when histamine is injected into a dog's skin, a method becomes available for the study of the mechanism of such lesions. It was previously reported that normal skin contains a substance which, when added to a given concentration of histamine, makes the wheal larger in comparison with the wheal produced by the same concentration of histamine without the added skin extract. This substance in the skin extract has now been found to be calcium. Calcium enhances the effect of histamine only when it is in a very dilute solution, in as small a concentration as ten parts per million. Highly concentrated solutions of calcium seem to reduce the size of the wheals. No report is yet available of clinical experiments following these observations. In the author's opinion the problem is one of securing a high concentration of calcium ions.

Sensitization.

M. B. SULZBERGER AND R. L. MAYER (*Archives of Dermatology and Syphilology*, October, 1931) have carried out experiments in the sensitization of guinea-pigs at Breslau and also at Zurich and New York. They come to the conclusion that one factor, hitherto regarded as constant, is responsible for most variable results, and that this factor is the geographical location of the investigations. This factor no doubt comprises regional, seasonal, dietary and other influences. Chance observation showed that guinea-pigs reacted differently when sensitization with the same brand of "Neoarsphenamine" was attempted in Breslau, Zurich and New York. In Breslau 98% of the animals became sensitized, and in New York none, whilst at Zurich about 54% reacted. A repetition of the experiments led to the same results. A study of the exact conditions influencing these variations may, in the opinion of the authors, lead to discoveries of practical importance in prophylaxis. In further experiments conducted at Breslau on the influence of diet, it was found that green fodder (alkaline ash) inhibits sensitization and that dry fodder favours it. This is considered not so much a question of vitamins as one of acid base balance. The authors refer to the recent work of Greenbaum on syphilitic infection in rabbits, in which the feeding of the rabbits influenced the percentage of "tabes."

X Ray Treatment of Psoriasis.

K. HOEDE (*Deutsche Medizinische Wochenschrift*, April 3, 1931) discusses the indications and contraindications to the use of X rays in psoriasis. He emphasizes that the skin erythema dose is not more than half the normal amount. Radiation should not be employed until other methods of treatment have had an extensive trial. Radiation is also contraindicated in the presence of any signs of previous radiation—pigmentation or dilated vessels. It should never be employed when the lesions are covered with clothing, especially in the region of the elbow. It may be used in cases involving the face, forearm, back of the hands and nails. Radiation of the scalp is dangerous because of the risk of baldness. Radiation of the thymus has been unsuccessful. Before commencing a course of treatment the patient should be warned of the risks, and it is better to have a written assurance that no previous attempts at radiation have been made.

Epithelioma of the Skin.

J. BELOT (*Journal de Radiologie et d'Electrologie*, July, 1931) during the last twenty-eight years has treated at the Broca Hospital and at the Saint-Louis Hospital approximately 12,000 epitheliomata of the skin. After this period he has decided there is no one method of treatment, but that the technique and the dose must be calculated for each individual case. With curetting and radiation the author scrapes away all the friable neoplastic tissue and permanently enucleates the peripheral "pearls" which form the most radio-resistant tissue. Objection has been raised to this method on account of the possibility of inoculating the tissue around the growth, but the writer states that in twenty-eight years he has treated large numbers of epitheliomata and has seen no sign of this infection of the nearby skin. He is also certain that the most useful dose of X radiation is that which is given at one sitting. The application administered is between 15 and 25 Holzknecht units applied over the whole of the curetted surface and to the surrounding skin for a distance of about seven millimetres. Only three-tenths of a millimetre of aluminium are used as filter. The author prefers the longer wave length for the treatment of these superficial lesions. Contrary indications to this mixed method mentioned are when the lesion is infiltrated below the limit of the skin, when the epithelioma shows very marked fungations, when it is badly infected or when the growth has recurred after previous radiotherapy. When using diathermy, the author prefers to destroy principally the proliferating edges, after which he irradiates the whole lesion. He concludes with the remark that on the judicious employment of radiation and its combination with curettage and in certain cases with electrolysis and electro-coagulation depends the final success in treatment of a great majority of cutaneous epitheliomata.

Cerebro-Spinal Fluid in Early and in Latent Syphilis.

H. H. HOPKINS (*Archives of Dermatology and Syphilology*, September, 1931) states that of 405 patients with early and latent syphilis whose cerebro-spinal fluid was normal at the first examination, definite neurosyphilis developed in 2·4% and questionable neurosyphilis in an additional 1·2%, whereas 96% remained well over periods ranging from two to ten years. This is contrasted with 15% of definite and 30% of questionable cases of clinical neurosyphilis which developed in a series of patients with early or late asymptomatic neurosyphilis. The types of neurosyphilis observed were relatively mild, being confined to lesions of the cranial nerves, with the exception of one patient in whom

syphilitic epilepsy developed. No instances of parenchymatous neurosyphilis were observed. The excellent prognostic import of the original failure of the cerebro-spinal fluid to react is in striking contrast with that of the "originally positive" cerebro-spinal fluid. Late neurosyphilis rarely develops in patients with a normal cerebro-spinal fluid in early syphilis unless they undergo a subsequent clinical or serological relapse. In patients with latent syphilis and a normal cerebro-spinal fluid, neurosyphilis in any form rarely if ever develops.

UROLOGY.

Massage of the Prostate.

A. L. WOLBARST (*Journal of Urology*, May, 1931) describes the apparatus he has devised for the pneumo-vibratory massage of the prostate. It consists of an electric pump capable of 8,000 revolutions per minute, which is connected by rubber tubing to a bulb placed on the pump of the index finger, both being enclosed in a finger cot. In using the method it is important that no pressure should be made, the vibrating bulb being merely held in contact with the prostate. The author claims that, as a substitute for digital massage, this method attains the same or better results without pain or disagreeable sensations to the patient, and with much less fatigue to the physician.

Cordotomy.

F. C. GRANT (*Journal of Urology*, June, 1931) draws attention to the advantages of cordotomy for intractable pain due to genito-urinary diseases and supports his arguments with abstracts of thirteen of Frazier's cases. He claims three advantages over other surgical procedures for relief of pain, namely: (i) Section of the spino-thalamic tract produces contralateral anæsthesia for pain up to one or two segments below the level of section; (ii) motor function, touch, position and muscle sense are unimpaired; (iii) only three spines and laminæ require to be removed. No details of technique are given, but the author states that section should be made at the fourth thoracic segment. All the patients were free from pain after operation and one patient had the second side operated upon. This patient is living after nine years, one patient survives after five years, five survived six months, three survived from three to five months, and the remainder died within a few days or weeks of operation. Nevertheless Grant considers that "the operation is sufficiently effective to warrant advising a patient to have it performed."

Bacillæmia in Urogenital Tuberculosis.

R. LICHTENSTERN (*Münchener Medizinische Wochenschrift*, March 20, 1931) points out the value of the examination of the blood for the tubercle bacillus in the subjects of genito-urinary tuberculosis. This is done by citrating eight to ten cubic centimetres of blood, hæmolysing it by 5% acetic acid, centrifuging and culturing the sediment on a special medium recommended by Löwenstein. The author points out how the earliest stages of genito-urinary tuberculosis are frequently accompanied by bacillæmia, the demonstration of which makes an early diagnosis possible. Further, bacillæmia in patients who have had operative treatment, is a sign of the persistence of the specific infection, and, finally, the freedom of the urine from tubercle bacilli no longer justifies the view that the patient is cured. This may be assumed only when the blood also is free.

Carcinoma of the Prostate Gland.

H. RUBRITIUS (*Wiener Medizinische Wochenschrift*, July 4, 1931) discusses the incidence of malignant disease in prostatic hypertrophy. He emphasizes the frequence of malignant changes in prostatic adenomata and advocates early operation as the only method of decreasing such rates. Hæmaturia and radiating pain are the most important signs of malignant disease. Surgical treatment is of little avail for complete cure, as most cases are not diagnosed until practically inoperable. Treatment by X rays has not proved as successful as with malignant growths elsewhere. Radium implantation through the bladder has been advocated, especially by American authorities, but the results so far have not been satisfactory. It is significant that most writers have aimed at prolonging life rather than at effecting a complete cure. Early prostatectomy at any age still remains the only safe prophylactic measure for malignant disease of the prostate.

Treatment of Impotence.

J. K. MAYER (*Deutsche Medizinische Wochenschrift*, August 14, 1931) discusses the various treatments adopted for masculine impotence. He considers that hormonal treatment and "Yohimbin" act merely as psychical agents. Apart from surgical measures, such as vaso-ligation, transplantation of testicles or section of the sympathetic fibres around the spermatic artery, he considers that an increased blood supply to the testis is best obtained by diathermy. He employs a current of 0·6 to 1·2 ampères. Each application lasts from fifteen to twenty minutes and is repeated every three days. Diathermy has been employed in twenty cases with complete success as regards erection and ejaculation. Its advantage in such cases lies in the simplicity and lack of complications following the treatment.

Trauma and Renal Calculus.

BLOCK (*Zeitschrift für Urologie*, May, 1931) reports a case in which, after a severe accident, the patient required prolonged catheterization and developed cystitis. After three months calculi were found radiographically in both kidneys, one being a staghorn calculus occupying the whole pelvis. Treatment consisted in the administration of large quantities of mineral waters. Eventually the patient commenced to pass small calculi and continued to do so until no further stone could be seen in the X ray picture. Block considers that post-traumatic calculus is laid down on a fibrin framework and is less dense than the ordinary laminated stone. Fragmentation, he believes, is encouraged by the administration of large quantities of mineral waters, and he regards those containing carbonates and silicates as the most valuable in this respect. Theoretically, only phosphatic or carbonate calculi can be "dissolved" in this way, and then only if their structure is very fragmentary. Uric acid and oxalate calculi cannot be dissolved in the kidney. The author emphasizes the necessity for accurate history taking and chemical analysis of calculi. When a stone is formed soon after trauma, the possibility of its dissolution by this means must be considered.

Hydatid Cyst of the Kidney.

L. A. SUBRACE AND H. MEZZERA (*Revista Medica Latino-Americana*, July, 1931) discuss the pyelographic diagnosis of hydatid cyst of the kidney. The diagnosis can be made with certainty when the cyst has opened into the urinary passages, because the pyelograph is then characteristic. The authors describe what they call "*El signo de la copa*" (the cup sign), which is pathognomonic. A shadow is then seen having a regular rounded contour in intimate contact with the renal pelvis and above all with a calyx. The shadow has a base like a lunar crescent, more or less open, supporting itself on one of the calyces. The crescent shaped base which supports the cyst, is formed exclusively of the cyst envelope and not by any enlargement of the calyx. The diagnosis is made from renal neoplasm because the latter deforms the pelvis and makes it irregular. The appearance seen is of a solid mass forming an integral part of the renal shape. The authors, who illustrate their article with pyelograms of some twelve cases, also describe the pyelographic appearances which will lead the diagnostician to suspect hydatid cyst when the cyst has not communicated with the urinary passages.

Pituitrin and Pyelography.

W. B. DRAPER, W. DARLEY AND J. L. HARVEY (*Journal of Urology*, July, 1931) show that intramuscularly administered pituitrin reduces the size of shadows produced by intravenous pyelography, the reduction in size being proportional to dosage. They conclude that this is due to increased peristalsis. A clinical case of bilateral infected hydronephrosis due to transverse myelitis is cited. In this patient the pain during attacks was relieved by pituitrin more efficiently than by morphine, and ultimately the attacks ceased, although the urine remained infected.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held in the Medical Society Hall, East Melbourne, on August 5, 1931, DR. H. DOUGLAS STEPHENS in the chair.

Scarlatina.

DR. F. V. G. SCHOLES, Medical Superintendent of the Queen's Memorial Infectious Diseases Hospital, Fairfield, Victoria, read a paper entitled "Scarlatina" (see page 703).

DR. H. MCLORINAN said that he had very little to add to what Dr. Scholes had said. The conception of scarlet fever as a streptococcal infection was important from the point of view of serum therapy and of bacteriology. It suggested among other things that puerperal infection should be treated by attacking the toxin rather than the streptococcus.

DR. JOHN DALE paid a tribute to the excellence of the paper. He envisaged the idea of mutation of strains and the nature of virulence as "the other side of the coin" from the idea of the resistance of the host to infection. The attainment of immunity might be an expression of an extensive symbiotic relationship between the invading organism and the host species. Doubtless more would be known of this problem before very long. Dr. Dale mentioned the possible influence of great changes in the diet of peoples on changes in the nature of the diseases affecting them. He questioned the authenticity of the immunity of the Eskimos to scarlet fever, and also doubted the value of the Dick test in communities where scarlet fever was not of frequent occurrence.

DR. J. H. SHAW expressed appreciation of the paper, especially in so far as it applied to his own special field, and said he was glad that Dr. Scholes approved of tonsillectomy in certain cases as a means of rendering the patient less subject to infection by diseases of the scarlet fever type. Writers in the American literature were sceptical of the value of tonsillectomy in preventing rheumatic fever, especially after a recrudescence. Concerning nasal infections, Dr. Shaw said that in all cases of sinusitis there was a high incidence of measles and scarlet fever in the patients' histories. Bacterial cultures from the post-nasal areas were unsatisfactory owing to the mixed flora. Persistent otorrhœa with deafness was common following scarlet fever, and, comparing this disease with mumps, he had found that deafness was more common following mumps, but otorrhœa more common after scarlet fever. He would like to hear the results of the recent efficient surgical treatment of ear, nose and throat conditions at Fairfield Hospital.

DR. F. NYULASY recorded his experience of treatment with scarlet fever antitoxin in the case of an only child who was very ill with scarlet fever. The child would take neither medicine nor nourishment, and seemed likely to die. A few hours after the administration of a therapeutic dose of scarlet fever antitoxin, the child was much improved and made a rapid and complete recovery.

DR. W. T. NELSON asked what relationship an epidemic of acute hæmorrhagic nephritis in young children might have to scarlet fever infection. Although the relationship had not been obvious, a bacteriological investigation of some epidemics of this type had revealed a long chain streptococcus of hæmolytic type as the causative organism.

DR. GUY SPRINGTHORPE asked two questions: First, was the presence of adenoids an important factor in the occurrence of late streptococcal infections in children following scarlet fever? Secondly, what were the factors predisposing to the development of the well known type of abnormality associated with enlarged tonsils and adenoids?

DR. A. P. DERHAM said that he did not think it fair for Dr. Springthorpe to ask his second question, as the answer, with advice as to prevention, could be obtained almost any day over the wireless, in the daily Press, and in a thousand and one pseudoscientific publications.

For his part he was also intensely interested in the questions raised in the paper. On his study table there always reposed Dr. Scholes's text book on diphtheria, measles and scarlatina, and he had come to look on this book as a veritable bible of authority. There was one point in treatment recommended in scarlet fever about which he would like to ask Dr. Scholes's latest opinion, and that was his advocacy of a protein-poor diet during the first three weeks of convalescence from scarlet fever. He (Dr. Derham) did not appreciate the theoretical basis for this restriction, and he wondered if Dr. Scholes still considered it justified by his practical experience.

DR. H. LAWRENCE STOKES asked, with regard to scarlet fever carriers, whether Dr. Scholes gave antitoxic serum to lessen infectivity and also whether he performed tonsillectomy for the same reason?

DR. R. H. SOUTHBY said, in regard to the isolation of streptococci from the urine, that in a series of cases so investigated, he had found pneumococci in the urine of some patients with pneumonia who had developed nephritis in the course of the disease. He would like to know if streptococci were found in the urine of patients developing a scarlatinal nephritis, and also whether the throats of scarlet fever patients were swabbed as a routine procedure at Fairfield Hospital.

DR. F. G. MORGAN said that, with regard to the development of a racial immunity and its experimental investigation, it was difficult to find animals susceptible to toxins affecting human beings, and he would be glad to hear Dr. Scholes's opinion regarding racial immunity among humans. He had had an interesting experience in early experiments with scarlet fever toxin. A resident medical officer at the Children's Hospital had been given 250 skin test doses of the toxin and had developed a high fever, a scarlet fever rash, and was in bed for two days. Animals, however, did not react even to five cubic centimetres of the toxin.

DR. HUGH G. MITCHELL asked whether Dr. Scholes had considered the question of a controlled observation of the incidence of scarlet fever in families where some children had had their tonsils removed and some had not.

DR. H. DOUGLAS STEPHENS expressed the thanks of the meeting for the paper and also for the interesting discussion. He was optimistic as to the value of scarlet fever antitoxic serum, and he asked whether it should be given in every case when the patient could afford it. He also asked Dr. Scholes's opinion as to the prophylactic value of the serum. Personally, he had had good results from its prophylactic use. He asked whether it was justifiable to use the serum in the treatment of chronic rhinorrhœa and otorrhœa or whether it would be better to use toxin-antitoxin or anatoxin in such cases. At the Foundling Hospital in East Melbourne there had been some cases of mild scarlet fever among the nurses and mothers, and Dr. Hilda Bull had found certain infants sensitive by the Dick test and gave them antitoxic serum. None of the infants developed scarlet fever, but streptococci were recovered from sores and noses.

In reply, Dr. Scholes said that he had no doubt that many of the worst cases of puerperal septicæmia were caused by hæmolytic streptococci which were probably identical with the *Streptococcus hæmolyticus* of scarlet fever and were communicated to the patient from the throat of one of the attendants. The scarlet fever streptococcus was, loosely, any streptococcus which could produce a toxin and an exa[illegible]hem. The streptococcus which caused erysipelas, probably could, in a suitable subject, cause scarlet fever, and *vice versa*.

It was proper to give antiscarlet fever serum as a prophylactic to any lying-in woman in whom puerperal sepsis was considered likely to occur. It was not fair to expect recovery in all cases treated therapeutically with the serum, as the placental site presented such a large open wound for the inroad of infection. The antibacterial streptococcal serum had not proved of any value in treatment.

Dr. Scholes thought Dr. Dale a little too pessimistic regarding the bacteriological position. It was true that they knew little of the inner life of bacteria, and little of

mutation, but knowledge was becoming clarified regarding the types of streptococci which could produce scarlet fever. It seemed that the organism which produced erysipelas in the age periods under six months and over fifty years, could in the intervening period produce scarlet fever.

Regarding the influence of diet and social condition on the incidence of scarlet fever, the Chinese and Annamites were practically immune from the disease. It had been suggested that dark skins had something to do with immunity to streptococci, but, on the other hand, the Japanese had much less immunity. It could not be suggested that the diet and hygienic conditions of the Chinese would tend to produce more immunity than that of the English in the middle of the last century.

The incidence of scarlet fever was greater in high altitudes than in low altitudes, in spite of the crowding of populations in the latter. At high altitudes in tropical countries also scarlet fever was ten times as prevalent as at sea level, and it was definitely a disease of temperate zones. Racial immunity could not be explained on any such simple basis, as the Eskimos had almost total immunity and did not contract scarlet fever even when employed as wardsmen in fever hospitals in temperate climates. In some tropical countries 50% of children were born "Dick positive," but did not get scarlet fever.

With regard to active immunization, valuable work had been carried out on a large scale in Soviet Russia. Large groups of up to eighty or ninety thousand children were divided into roughly equal sections, half being immunized and half used as controls, in periods just before epidemics were expected.

One group of 5,000 was immunized with toxin, one with toxin combined with vaccine, and a third group with anatoxin. There was not much difference in the scarlet fever incidence in these three groups, but there had been an astounding drop in the incidence and mortality in all three groups compared with the control groups, the case mortality being from 5% to 10%, as compared with 30% in the controls.

Dr. Scholes said that while the Health Commission was sitting some years ago he had given evidence suggesting the application of the Dick test to all women in the later months of pregnancy and their active immunization with scarlet fever toxin. The general public were not sufficiently afraid of scarlet fever to submit to general immunization.

In reply to Dr. Shaw as to the results of tonsillectomy in cases of rheumatic fever, Dr. Scholes said that the bad results reported by some observers should be qualified. In patients with unhealthy tonsils, tonsillectomy should be performed before the first attack of rheumatic fever, as after an attack the results were much less satisfactory.

In scarlet fever tonsillectomy during the second week of the disease should be a valuable prophylactic against the rheumatic complications. With regard to rhinorrhœa following scarlet fever, there was some argument in favour of tonsillectomy before this occurred, as after its appearance the sinus infection was not much affected by tonsillectomy. Regarding the removal of tonsils and adenoids in the treatment of otorrhœa following scarlet fever, there was not sufficient evidence to enable him to speak with authority of the results, but in many cases the operation had been followed by rapid recovery of the ear condition. Dr. Scholes, continuing, said that deafness did not very often follow the otitis of scarlet fever. In one of his early cases at Fairfield the ossicles of both ears came away with the discharge, and the patient left hospital not very deaf.

In answer to the question whether antitoxin should be given in mild cases as well as in severe cases, Dr. Scholes said that it certainly should be given in severe cases, and that in mild cases in private houses he suggested its use unless the expense was prohibitive. In infectious diseases hospitals he doubted its value in mild cases, because relapses and reinfections were so common that the use of serum seemed hardly worth while. In private houses it should be given in every case except the very mild case in which it did not seem likely that even desquamation would occur. The purpose was twofold: first, to neutralize toxin and, secondly, to abort the enanthem. The throat cleared rapidly and the tongue did not even peel. If given at all in such very mild cases, it would be probably of most value at the end of the third week, when it would tend to prevent kidney and gland complications.

He considered that the epidemic of acute hæmorrhagic nephritis mentioned by Dr. Nelson might have been caused by the scarlet fever organism, as, in some recently reported epidemics of this type, hæmolytic streptococci were causative.

In answer to Dr. Guy Springthorpe's questions, he (Dr. Scholes) considered it was wise to remove infected adenoids, whether the tonsils were removed or not. Regarding the factors predisposing to unhealthy tonsils and adenoids, he was unable to give a satisfactory answer. Twenty years ago he had read a paper on scarlet fever before the same Branch, and had asked whether any of the older practitioners present had noticed any change in the incidence of unhealthy tonsils and adenoids in the preceding fifty years. No definite opinions were obtained.

The problem was important, because the prevention of scarlet fever lay in finding out what were the causes so predisposing. Was the tonsil disease due to infection of previously healthy tonsils or to infection of tonsils previously rendered unhealthy by some other influence?

The diphtheria carrier condition did sometimes clear up after the hypodermic administration of five to 10,000 units of diphtheria antitoxin. Dr. McLorinan had proved this in a number of cases. They were trying the same procedure in certain carriers of scarlatina. In patients with mild scarlatina who could be sent home early (within fourteen days), it was best to withhold antitoxin until just before they left hospital.

In reply to Dr. Southby, Dr. Scholes said that there had not been any thorough investigation at Fairfield of the bacteriology of the urine in scarlet fever nephritis. It was generally found that streptococci were not found in the urine in these cases and it had come to be considered that scarlatinal nephritis was purely toxic. The matter, however, was worthy of fuller investigation, and it was certain that scarlatinal nephritis did sometimes predispose to nephritis later on. This predisposing attack was not usually of the hæmorrhagic or glomerular type, but rather the interstitial form occurring as a later complication or sequela.

The early hæmorrhagic type did sometimes lead to chronic nephritis, especially with recurring attacks of subacute symptoms every few months, to reappear, perhaps, three or four years later as a fatal chronic nephritis. The swabbing of suspected throats and the throats of all scarlatinal patients for hæmolytic streptococci at Fairfield Hospital was usually a routine procedure, but recently had been less uniformly done owing to overcrowding.

A valuable investigation had been carried out on this point in Japan. When patients were discharged from a fever hospital without swabbing, a certain percentage of family infections occurred, but none occurred in groups which were proved free from infection by swabbing before discharge.

In reply to Dr. Derham, Dr. Scholes said that he did not now restrict the diet during convalescence quite so rigidly as indicated in his text book, but it was desirable to exclude irritating substances with little food value, such as alcohol, condiments, rich roast meats *et cetera*. It was not necessary to withhold eggs, fish and other nourishing food.

In reply to Dr. Morgan regarding racial immunity, Dr. Scholes said that dark skinned races were almost immune to scarlet fever, though hæmolytic streptococci were found in their throats; but they did not get scarlet fever if they went to cold climates.

In the United States of America rheumatic patients harbouring hæmolytic streptococci were sent to Porto Rico, where they recovered. The local inhabitants did not get rheumatism.

Antitoxin or toxins from certain old recultivated strains of streptococci were found to be of no value in producing passive or active immunity to scarlet fever. This fact might have some relation to the problem of mutation of streptococci. In some patients suffering from scarlet fever and living in two Continental hospitals under observation,

all the hæmolytic streptococci in their throats changed after some weeks to *Streptococcus viridans*, and the reverse had occurred in certain culture media.

Scarlet fever was a comparatively rare disease in children who had had their tonsils removed, and in children who had previously had reasonably healthy tonsils. Very few children were admitted to Fairfield Hospital suffering from scarlet fever who had had their tonsils completely removed.

Statistics in America bearing on the desirability of removing tonsils and adenoids were misleading and contradictory. According to some observers, their removal seemed to have no beneficial effect in preventing pneumonia, colds, sinusitis *et cetera*. In fact, some figures indicated a reverse influence. No effect was produced on the incidence of measles, naturally. The best controlled investigations on the point, however, indicated that the removal of tonsils and adenoids had a beneficial effect on the incidence of scarlet fever, colds, rheumatism and diphtheria.

In reply to Dr. Stephens, he (Dr. Scholes) considered it wise to give prophylactic antitoxic serum to contacts in private houses, as active immunization was too slow a process.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at Warragul on July 18, 1931.

Achlorhydric Dyspepsia.

DR. J. M. ANDREW read a paper entitled: "Achlorhydric Dyspepsia." This paper was published together with the discussion in the issue of November 21, 1931.

Surgical Reminiscences.

DR. G. A. HAGENAUER read a paper in which he described surgical methods in vogue thirty-five years ago. He began by referring to the work of Professor Watson, formerly of the University of Adelaide, to his uncanny knowledge of surgical anatomy and to his demonstrations to Melbourne surgeons in the *post mortem* room at the Melbourne Hospital. Dr. Hagenauer said that in those days there were but few private hospitals in Melbourne and that surgical work was confined to a few men. At the present time Melbourne was overrun with private hospitals, and many surgeons were doing major surgical operations and doing them well. Dr. Hagenauer went on to describe the work at Saint Vincent's Hospital, and referred particularly to the days set apart for the removal of tonsils and adenoids. There being no room in the wards, it had been necessary to accommodate as many as twenty patients on lounges along the verandas until they were able to return home after a couple of hours' rest. In those days the resident medical officer had no need to worry about secondary hæmorrhage, as dissection of tonsils was not done. Dr. Hagenauer said that about two years ago he had been nearly torn to pieces at a Branch discussion because he expressed the opinion that dissection of tonsils would go out of fashion on account of the risk of fatal hæmorrhage, and that the guillotine would again come into its own. It had been interesting to him to hear from a colleague recently returned from abroad, that in the great clinics in America, Germany, France and Great Britain the guillotine was being used for almost every patient up to the age of twelve years, and that dissection was reserved for adults and special cases.

Dr. Hagenauer went on to refer to the equipment of operating theatres of thirty-five years ago, to the sea-sponges, to the use of antiseptics such as carbolic acid and perchloride and biniodide of mercury, and to the use of silk for anastomosis of the bowel. He also referred to the use of kangaroo tendon, and said that he had found, when he settled in Gippsland, that wallaby tendons were much longer and of more uniform thickness than those of the kangaroo. Tuberculous enlargement of the cervical glands had been very prevalent, but their place was taken today by appendicitis. He found that when operating on a suppurating case of appendicitis the examining finger should be replaced by a drainage tube as soon as pus was located. It was astonishing how well the patient did in these circumstances. Only harm could be done if they burrowed about among the coils of intestine breaking down protective adhesions.

Dr. Hagenauer then described the advances which had been made in surgery of the prostate gland, of the antrum, of the breast and gall-bladder. He described the methods used in various types of gynæcological operation, and referred to the work of Howard Kelly, to whom a great advance in operative technique was due. Looking back over the years, he felt that they should appreciate the work of surgeons who had not enjoyed the present-day facilities. Perhaps in thirty-five years' time it would be found that the scalpel had in a measure, at least for malignant growths, been supplanted by radium or some other scientific discovery. Surgeons then might express similar views in regard to the surgeons of today.

Perthes's Disease.

DR. CONRAD LEY showed a male patient, aged nine years, who was suffering from Perthes's disease. He was first seen on October 18, 1928, when he complained of limp involving the right leg and also of pain in the right knee joint. There was no previous history of injury. On examination, the boy was very pale in appearance and poorly nourished. He had no fever. There was obvious wasting of the right thigh, but no shortening and but slight limitation of rotation and of abduction. X ray examination revealed fragmentation of the head and broadening of the neck of the femur.

The patient was protected from weight-bearing by being placed in a frame for eighteen months. Serial pictures taken at intervals during this time showed a slow regeneration of bone. He had been back to normal joint activity for six months. Examination at the time of the meeting showed evidence of slight wasting, no limitation of movement and about eighteen millimetres (three-quarters of an inch) of shortening, due to a slight degree of *coxa vara*.

DR. H. DOUGLAS STEPHENS said that the absence of history of injury was unusual; possibly the injury only called attention to the condition. When this history, with the limp and pain referred to the knee, in conjunction with the X ray findings, was considered at present, three years after the onset, the diagnosis was between an arthritis of some type or Perthes's disease. He would exclude a slipped epiphysis on the skiagram alone. The subsequent history confirmed the diagnosis of Perthes's disease and the 1·25 centimetres (half inch) of shortening was further confirmation, the slight shortening being due, not to the alteration in the angle of the neck, but to the flattening of the head. The only point that raised a doubt in his mind was the pale appearance of the boy, and he wondered whether there had been any enlargement of the spleen or whether a Wassermann test had been made. He considered the result was very good, and congratulated Dr. Ley on it. Although some authorities thought that the child should be encouraged to use the limb and allowed it to bear full weight, he took the weight off the hip and agreed with Dr. Ley in that regard.

Asked by Dr. Ley whether there was any relation to congenital dislocation of the hip, Dr. Stephens stated that he did not think there was any relationship or that the typical appearance was present, although in some cases of congenital dislocation of the hip in which there had been difficulty in manipulation, mushrooming from injury to the epiphysis might occur.

DR. MERVYN STEWART said that he would like to hear an exposition by Dr. Colin MacDonald on the radiological appearance of osteochondritis. He agreed with Dr. Stephens that he had never seen a case without a history of some injury. As regards treatment, he thought that the weight should be taken off the hip.

The shortening was due to the *coxa plana* or shortening of the neck and the flattening of the head, and not to any alteration in the angle of the neck. There were two types, one with a globular head and one with a flattened head with more fragmentation of the epiphysis, which usually gave rise to greater disability.

He thought the result in this case was very good. In addition to watching the opposite hip, the affected hip

should be watched for the possibility of osteochondritis developing at a later stage.

DR. H. FLECKER stated that the X ray appearance was typical of Perthes's disease with fragmentation of the epiphysis, shortening of the neck and flattening of the head of the bone. The clinical history also was typical. The prognosis was favourable and he had not yet seen any serious results follow, although it was questionable whether the condition of some of the patients developing *coxa vara* or osteoarthritis at about sixty years of age had not started early in life as Perthes's disease. Time was the only factor that would allow a decision to be made.

DR. DOUGLAS THOMAS, speaking on the point of the subsequent development of a *coxa vara*, did think that there was a relationship with Perthes's disease, which was probably the starting point of the disease. He instanced the case of a woman, aged thirty, whom he had recently seen, who had developed a *coxa vara* with early osteoarthritis, which he was inclined to look on as an osteoarthritis secondary to old Perthes's disease.

Injury to Elbow and Ulnar Nerve Paralysis.

Dr. Ley's second patient was a girl, aged fourteen years, who was first seen on December 12, 1930, with a history of injury to the left elbow joint, sustained two months previously. Examination at that time showed the joint ankylosed at a right angle; there was also present a partial ulnar nerve paralysis. X ray examination of the joint revealed a fragment of bone locked in the elbow joint on the medial side, apparently separated from the medial epicondyle of the humerus.

At operation the joint was opened and the loose fragment removed. This immediately allowed almost full extension of elbow joint. The limb was then put up in a light plaster cast for three weeks, active movements being commenced about ten days after operation. Following operation there was an immediate improvement in ulnar nerve function, the area of skin anæsthesia being diminished. At the time of the meeting, about seven months after operation, there was a good functioning joint with full range of movement. The ulnar nerve, however, had failed to recover completely.

DR. V. HURLEY said he had seen two or three cases with a chip off the medial condyle where it was in relation to the nerve, and the chip had been carried into the joint. The joint disability was purely mechanical from the presence of the foreign body. The nerve injury was indirect and due to dragging on the nerve.

The treatment was operative and the best method of approach was that which had been adopted where an incision had been made on to the fragment from the medial side. The detached piece of bone was practically subcutaneous and it could be lifted out of the joint and replaced in its normal position. Usually there was complete recovery both of movement of the elbow and of the nerve; but in this instance there was an unusually long time before operation, and therefore he would not expect such a good result. Platt, of Manchester, quoted similar cases with a good recovery.

DR. A. HAILES disagreed with Dr. Hurley, as he did not think a good recovery would occur. In the only cases he had seen which had done well, the interossei and lumbricals had recovered in three months; but in this instance there was no recovery after a longer period, and he did not think there would be further improvement.

He instanced the cases of trauma to the nerve as it lay behind the medial condyle, in which transposition of the nerve had been done. In such cases, if there was no improvement in four months, none would occur.

DR. JOHN KENNEDY said he did not agree with Dr. Hailes, and he had seen improvement occur up to twelve months. In this case there was not much pull in the small muscles of the hand; but he considered that they would improve.

DR. MERVYN STEWART said he wished to congratulate Dr. Ley on his result. He would expect further improvement. He referred to cases of ulnar paralysis associated with *cubitus valgus* in which he had transposed the nerve. He had shown them previously to illustrate Adson's technique, which he used; but he wanted to refer to one on which he had looked with a hopeless feeling, but in which there was ultimately complete recovery. In this case there had been complete reaction of degeneration twelve months after injury, and he had only transposed the nerve. He did not splint the hand, as he could not devise any adequate splint.

In the light of the case he was referring to, he would expect Dr. Ley's patient to improve; but if there was no further improvement, he would suggest transposition of the nerve.

A Septic Hand.

Dr. Ley also showed a male patient, aged forty years, who had suffered from acute suppurative tenosynovitis of the *flexor pollicis longus* muscle. The patient was first seen two and a half years before with a small infective focus in the ball of the right thumb, said to have been due to a small thorn. Incision at this time liberated a small quantity of pus. Next day the patient's temperature went up to 40° C. (104° F.) and he had a severe rigor. Accompanying this there was tenderness extending down along the course of the tendon of the *flexor pollicis longus* muscle. An incision was made on to the tendon. The sheath was found filled with pus which extended into the forearm. Radial and ulnar incisions were made and a small drain was passed under the tendons. Next day tenderness and swelling had passed further up the forearm. Two more lateral incisions were made over the most tender points; this resulted in pus being liberated from the space over the interosseous membrane. About this time two severe hæmorrhages occurred from the radial artery. These were controlled by forceps, which were left *in situ*. At this juncture, about twelve days after the onset, the patient was in great danger of losing his life and had septicæmia; the forearm was practically bathed in pus, two severe hæmorrhages occurred, and in addition septic bronchopneumonia supervened. Gradually the patient's condition improved and he settled down to a slow recovery. Many superficial pockets of pus had to be opened up. One week after onset his tendon sheath of the little finger and ulnar bursa had to be opened up. Three weeks after onset 5·0 to 7·5 centimetres (two to three inches) of his *flexor pollicis longus* sloughed out, and sloughing of the flexor tendons to the index and pleximeter fingers followed. Two months after the onset all suppuration had ceased. During the whole time the hand was kept in the position of function. At this time the radial artery was obliterated, slight wrist joint movement was present, and the loss of the *flexor pollicis longus* was balanced by the action of the *flexor pollicis brevis* and the *adductor pollicis*. The index and middle fingers were flail-like in action. No interphalangeal flexion was present, but only metacarpophalangeal flexion. Active movements were then encouraged. Great improvement had taken place in the development of collateral circulation and in movements. The hand at the time of the meeting was quite a useful one, except for the two flail fingers, due to the loss of the *flexor digitorum profundus* tendons.

DR. JOHN KENNEDY said that the little and ring fingers were good, and for the index and middle fingers the tendons could be felt pulling in the scar in the palm. He had a good grip with his little finger and an astonishingly good grip with his thumb, using the *opponens*. There was no hope of "getting a new tendon with the sheath of tendons to index destroyed"; but by ankylosing the interphalangeal joints at rather less than semiflexion one could get good gripping power in these fingers from the action of the short muscles on the first phalanx.

DR. A. HAILES said that possibly over a very long period it might be possible to do something with the fingers; but it would take years, and he did not think the effort would be worth while.

DR. V. HURLEY thought there was no hope of tendon movement, and he himself had no experience of fixation as Dr. Kennedy had suggested. He thought the patient had astonishingly good function.

DR. J. NEWMAN MORRIS said he thought the case a good illustration of Kanavel's description of infection, and was of interest as showing what happened if they did not deal

quickly and adequately with any infection. As regards prognosis, he thought there was very small expectation of results from tendon transplantation, certainly not more than 10% improvement. He thought Dr. Kennedy's suggestion was worth consideration.

Osteomyelitis and Acute Tuberculous Bronchopneumonia.

Dr. Ley also showed a boy, aged fifteen years, who was suffering from acute tuberculous bronchopneumonia. The patient was quite well till four years ago, when he was admitted to hospital with acute osteomyelitis of the right tibia. There was an extensive involvement of the shaft, which was treated radically. He very nearly succumbed during the acute stages of the disease. He made a slow recovery and had a long stay in hospital. He was readmitted several times later for removal of small sequestra and skin grafting. He then put on weight and appeared to be doing quite well until about three months ago, when he commenced to go down hill, his symptoms being loss of weight, loss of appetite, persistent cough and hoarseness. He was then readmitted to hospital. Examination at the time of the meeting revealed an obviously wasted and sick patient with a high, swinging temperature and rapid pulse. Physical examination revealed diffuse signs of a bilateral bronchopneumonia. The X ray picture as shown demonstrated a very thick sowing of miliary tubercles through both lungs. The second sputum examination revealed numerous tubercle bacilli.

DR. D. M. SILBERBERG thought that the chest signs, the chart, the gradual onset and X ray findings combined to suggest pulmonary miliary tuberculosis. It was a truism not to be put off by one "negative" sputum. He had seen cases in which the sputum had been "negative" for a long time. The treatment must be along general lines. "Ostelin" combined with calcium would be useful. He had had no personal experience with gold salts.

DR. L. HURLEY agreed with Dr. Silberberg. He also thought that if the condition were not tuberculous, the patient would not look so well. The X ray appearance was exactly similar to two cases that he had seen in which tuberculosis had been the diagnosis. The diffuse mottling was typical. He had used gold injections in six cases and was not impressed by the result. There was diminished sputum for a time, but the improvement was only temporary.

DR. H. FLECKER said he considered the skiagram of the chest was typical of tuberculosis, but the skiagram of the tibia did not suggest tuberculosis.

DR. CHARLES SUTHERLAND also thought a von Pirquet test might be of value. He thought that probably the test was underrated, even though it might be "negative" in an acute case.

DR. MERVYN STEWART said lung infection was often associated with a suppurative bone condition; but in his experience they usually appeared in a few days—they were acute conditions. So much was he impressed by their association that he had learned not to operate if there were lung signs or a pericardial rub.

Multiple Fractures.

Dr. Ley's last patient was a man, aged twenty-eight years. The patient was admitted two and a half months ago, having been badly smashed in a motor cycle accident. He had compound fracture of the femur in the middle third and simple fracture of the tibia and fibula of the same leg in the middle third. In addition to the injuries named, he had extensive scalp and facial lacerations.

Under general anæsthesia his facial injuries were repaired and his right leg, after reduction of the fractures was placed in a straight Thomas splint. X ray examination next day revealed still great displacement, the upper femoral fragment being flexed and the lower one displaced backwards. He was then transferred to Hamilton Russell's apparatus after better reduction was effected. This then led to trouble with the lower fracture, which hitherto had assumed a good position. The patient was then put on a Thomas splint bent at the knee, the upper fracture being controlled by calipers inserted into the condyles and the lower one by extension with the Sinclair skate and glue. The patient's condition then settled down and went ahead quite satisfactorily. All apparatus was removed two months after injury; good firm union occurred in the upper fracture; slight mobility was present in the lower fracture, which necessitated support, with a small plaster cast. No shortening was present.

The point of interest in this case was the difficulty experienced in treatment of double fracture of the same leg, also the great comfort derived from the use of calipers for the femoral fracture and the Sinclair skate extension for the lower fracture.

Duodenal Ulcer.

DR. R. D. SMITH showed a man, aged thirty-eight years, who was suffering from a duodenal ulcer. The patient was quite well till nine years ago. He then commenced to get attacks of pain in the upper part of the abdomen and had had the pain off and on ever since. The first attack was preceded by slight indigestion; the attack came on suddenly and the pain was acute. He was taken to hospital, where he spent three days, after which he returned to work. Since then he had never been really well and suffered from some degree of indigestion and had periodical attacks of pain in the upper part of the abdomen, accompanied by vomiting. About one month before the meeting he had an attack of this kind. On examination he looked thin and pale, the heart and lungs were normal. No abdominal rigidity was present, but slight tenderness was noted in the mid-line, about midway between the xiphisternum and the umbilicus. There was no enlargement of the liver or spleen. No mass was detected. The tongue was furred and the breath offensive. X ray examination yielded findings suggestive of duodenal ulcer.

DR. W. W. S. JOHNSTON said the possibility of cholecystitis had to be considered and should probably be investigated first. Duodenal ulcer might possibly be definitely revealed by X rays. He thought that the fæces should be tested for occult blood to get more definite evidence of hæmorrhage, and a fractional test meal would also give evidence of value. Treatment was undoubtedly rest in bed, strict diet, fluid and gradual "working up." If no response occurred in one month, operation would then have to be considered. This suggestion for treatment depended on a gall-bladder lesion being excluded.

DR. IVAN MAXWELL agreed with Dr. Johnston, but said he would like to emphasize the need for intense alkaline treatment. Alkali should be given every two hours with milk diet, and the patient should be in bed for one month at least.

DR. L. HURLEY said that he did not regard the diagnosis as established. The history was very unconvincing. Deformity of the duodenal cap was a very definite trap. He thought that gastric causes should be excluded. He found that the patient's knee jerks were present; but it was the only test he had carried out and he thought a complete investigation, including a Wassermann test, should be carried out. The chances were that if an exploration were done, nothing would be found, as was the usual happening if the findings were not typical. Intense ulcer treatment was the first requirement. As to the time in bed MacLean mentioned ten days, and that seemed to be about right.

DR. J. P. MAJOR agreed with the other speakers. If further investigation proved the presence of duodenal ulcer, then efficient medical treatment was the first essential. Some duodenal ulcers would not improve. He would stress the fact that this patient smoked heavily and his smoking had never been cut out. He thought that in these cases smoking must be stopped, as it definitely increased acidity. If treatment failed, then exploration should be undertaken.

DR. V. HURLEY agreed that the appearance of the duodenal cap was a definite trap. Posterior ulcer was a difficult problem of diagnosis. He found that he came more and more to rely on the history. He thought alkalis should be given a trial first.

NOMINATIONS AND ELECTIONS.

THE undermentioned has been nominated for election as a member of the New South Wales Branch of the British Medical Association:

Uther, Frederick Bryant, M.B., B.S., 1928 (Univ. Sydney), 8, Devonshire Street, Chatswood.

NOTICE.

THE Medical Secretary of the New South Wales Branch of the British Medical Association has forwarded the following list of books added to the library of the New South Wales Branch:

"The Œdema of Bright's Disease," C. Achard (Kegan Paul); "Insomnia," H. Crichton-Miller (Edward Arnold); "Lateral Curvature of the Spine," Lovett (P. Blakiston); "Laboratory Diagnosis," Osgood and Haskins (P. Blakiston); "Stepping Stones to Surgery," L. B. Rawling (H. K. Lewis); "Diseases of the Tongue," Spencer and Cade (H. K. Lewis); "Surgery: Its Principles and Practice," A. P. C. Ashhurst (Lea and Febiger); "Obstetrics and Gynæcology" ("Practical Medicine Series," 1930) (Year Book Publications); "Restoration Exercises for Women," E. A. Hornibrook (W. Heinemann); "Manual of Tuberculosis for Nurses," E. A. Underwood (E. S. Livingstone); "Ultra-Violet Radiotherapy," W. K. Russell (Jonathan Cape); "The Diabetic Life," R. D. Lawrence (J. and A. Churchill); "How to Keep Fit After Forty," R. Thornhill (Methuen and Company); "Happy Motherhood," P. W. Yeomans (W. Heinemann); "Climate and Acclimatization," A. Castellani (J. Bale, Sons and Danielsson); "Handbook of Therapeutics," D. Campbell (E. and S. Livingstone); "Operative Obstetrics on the Manikin," C. B. Reed (P. Blakiston); "The Collected Papers of the Mayo Clinic for 1930."

SCHOLARSHIPS AND GRANTS IN AID OF SCIENTIFIC RESEARCH.

Scholarships.

THE Council of the British Medical Association is prepared to receive applications for research scholarships as follows:

An Ernest Hart Memorial Scholarship, of the value of £200 *per annum*.

Three Research Scholarships, each of the value of £150 *per annum*.

These scholarships are given to candidates whom the Science Committee of the Association recommends as qualified to undertake research in any subject (including State medicine) relating to the causation, prevention or treatment of disease.

Each scholarship is tenable for one year, commencing on October 1, 1932. A scholar may be reappointed for not more than two additional terms. A scholar is not necessarily required to devote the whole of his or her time to the work of research, but may hold a junior appointment at a university, medical school or hospital, provided the duties of such appointment do not interfere with his work as a scholar.

Grants.

The Council of the British Medical Association is also prepared to receive applications for grants for the assistance of research into the causation, treatment or prevention of disease. Preference will be given, other things being equal, to members of the medical profession and to applicants who propose as subjects of investigation problems directly related to practical medicine.

Conditions of Award: Applications.

A copy of the regulations relative to the award of the scholarships and grants for 1932, and of the prescribed application form can be obtained on application to the Secretary of the Federal Committee of the British Medical Association in Australia, British Medical Association House, 135, Macquarie Street, Sydney. The completed application form is required to be submitted to the Secretary of the Federal Committee not later than March 5, 1932.

Applicants are required to furnish the names of three referees who are competent to speak as to their capacity for the research contemplated, to whom reference may be made.

Obituary.

DAVID FRANCIS BLANCHARD.

WE regret to announce the death of Dr. David Francis Blanchard, which occurred at Manly, New South Wales, on November 20, 1931.

PATRICK CHARLES HIGGINS.

WE regret to announce the death of Dr. Patrick Charles Higgins, which occurred at Brisbane, Queensland, on November 20, 1931.

Correspondence.

TRACHOMA AND DIET.

SIR: On reading Dr. Harvey Sutton's valuable Anne MacKenzie Oration in your issue of November 14, I was glad to learn that he and his Child Welfare Department had come to the conclusion that diet deficiency contributed to the persistence of trachoma.

I learn now from Dr. Madden's letter of November 21 that as early as 1928 Dr. Harvey Sutton and he had published the opinion that "there appears to be a large vitamin element entering the problem" of treatment.

My letter in your issue of October 10 was a result of the constant search of those of us engaged in ophthalmology for a missing factor in the induction of trachoma. Sir James Barrett's question to that effect in your journal was the immediate cause of my letter. I was gratified to find afterwards that he was enamoured of my suggestion. It is more particular and exact than Dr. Madden has quite gathered. I hope that we may come to recognize that deficiency in milk, butter and green vegetables—really in vitamin *A*—is a condition necessary to the establishment of trachoma, as it has been proved to be of keratomalacia and of xerosis. Infection from flies and from what they carry, though causing conjunctivitis, and at times very serious attacks, will probably not lead to the development of trachoma unless the soil has been prepared, especially in children, by this food deficiency.

Had I been conscious of Dr. Harvey Sutton's and of Dr. Madden's conclusions before writing my letter, I would have been only too glad to recognize them. It is, of course, possible that their views had come to my subconscious knowledge and helped to incubate the thought in my letter. I attributed it to McCollum's and McCarrison's work.

It is gratifying to know that Dr. Harvey Sutton and his colleagues will do what they can to institute prophylaxis in children by preaching the value of vitamin sufficiency, as his address does so admirably.

I should be glad to have the reprint of Dr. Madden's paper, which he so kindly offers.

Yours, etc.,

J. LOCKHART GIBSON.

Wickham Terrace,
Brisbane,
November 26, 1931.

Honours.

DR. A. R. McLEOD, of Sydney, has been invested with the Cross of Commander of the Order of the Hospital of Saint John of Jerusalem.

Books Received.

ELEMENTARY HISTOLOGICAL TECHNIQUE FOR ANIMAL AND PLANT TISSUES, by J. T. Holder, F.R.M.S.; 1931. London: J. and A. Churchill. Demy 8vo., pp. 111, with 23 illustrations. Price: 7s. 6d. net.

AIDS TO SURGICAL ANATOMY, by R. H. Hunter, M.D., M.Ch., Ph.D.; 1931. London: Baillière, Tindall and Cox. Foolscap 8vo., pp. 184, with illustrations. Price: 3s. 6d. net.

AIDS TO PHYSIOLOGY, by H. Dreyerre, Ph.D., M.R.C.S., L.R.C.P., F.R.S.E.; 1931. London: Baillière, Tindall and Cox. Foolscap 8vo., pp. 262, with illustrations. Price: 3s. 6d. net.

MINOR SURGERY, by L. R. Fifield, F.R.C.S.; Second Edition, revised by R. J. McNeill Love, M.S., F.R.C.S.; 1931. London: H. K. Lewis. Crown 8vo., pp. 447, with 281 illustrations. Price: 12s. 6d. net.

THE DIAGNOSIS OF NERVOUS DISEASES, by Sir James Purves Stewart, K.C.M.G., C.B., M.D., F.R.C.P.; Seventh Edition; 1931. London: Edward Arnold. Royal 8vo., pp. 738, with illustrations. Price: 35s. net.

BAINBRIDGE AND MENZIES' ESSENTIALS OF PHYSIOLOGY; Seventh Edition, edited and revised by H. Hartridge, M.A., M.D., Sc.D., M.R.C.P., F.R.S.; 1931. London: Longmans, Green and Company, Limited. Demy 8vo., pp. 583, with illustrations. Price: 14s. net.

A MANUAL OF GENERAL MEDICAL PRACTICE, by W. S. Sykes, M.A., M.B., B.Ch., D.P.H., M.R.C.S., L.R.C.P.; Second Edition; 1931. London: H. K. Lewis. Crown 8vo., pp. 225. Price: 7s. 6d. net.

DISEASES OF THE STOMACH, by H. Morton; 1931. London: Edward Arnold. Demy 8vo., pp. 191, with illustrations. Price: 10s. 6d. net.

OPERATIVE DENTAL SURGERY, by J. B. Parfitt, L.R.C.P., M.R.C.S., L.D.S.; Third Edition; 1931. London: Edward Arnold. Demy 8vo., pp. 412, with illustrations. Price 21s. net.

THE RHEUMATIC INFECTION IN CHILDHOOD, by L. Findlay, M.D., D.Sc., M.R.C.P.; 1931. London: Edward Arnold. Demy 8vo., pp. 193, with illustrations. Price: 10s. 6d. net.

EMERGENCY SURGERY, Volume II: Thorax, Spine, Head, Neck, Extremities, etc., by Hamilton Bailey, F.R.C.S.; 1931. Bristol: John Wright and Sons, Limited. Royal 8vo., pp. 432, with 430 illustrations, some of which are in colour. Price: 25s. net.

Diary for the Month.

DEC. 8.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
DEC. 10.—New South Wales Branch, B.M.A.: Branch.
DEC. 11.—Queensland Branch, B.M.A.: Branch (Annual).
DEC. 15.—New South Wales Branch, B.M.A.: Medical Politics Committee.
DEC 18.—Queensland Branch, B.M.A.: Council.

Medical Appointments.

Dr. C. J. Logan has been appointed Medical Officer of Health by the Meckering Road Board, Western Australia, pursuant to the provisions of *The Health Act*, 1911-1919.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xvi.

INSPECTOR-GENERAL OF HOSPITALS, ADELAIDE, SOUTH AUSTRALIA: Surgical Registrar.

THE HORNSBY AND DISTRICT HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Officers.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members desiring to accept appointment in ANY COUNTRY HOSPITAL, are advised to submit a copy of their agreement to the Council before signing, in their own interests. Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Mines. Toowoomba Associated Friendly Societies' Medical Institute.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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